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PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

JOHN NACHBAR, M.A., M.D.

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PART III

ODONTOLOGICAL SECTION
OTOLOGICAL SECTION
PATHOLOGICAL SECTION
SURGICAL SECTION
THERAPEUTICAL AND PHARMACOLOGICAL SECTION

INDEX

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PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE

ODONTOLOGICAL SECTION.

CONTENTS.

October 28, 1907.

PAGE

Presidential Address. By J. Howard Mummery, M.R.C.S., L.D.S. 1

The Teeth of Fossil Fishes. By John Humphreys, M.D.S.(Birm.) 7

November 25, 1907.

Some Notes on the Growth of the Jaws. By James T. Carter, L.D.S. 17

The Effects of Chronic Suppuration in the Molar of a Horse. By J. G. Turner, F.R.C.S. 26

January 27, 1908.

Specimens Added to the Museum. By J. F. Colyer (Honorary Curator) ... 27

Notes on a Case of Extensive Necrosis of the Mandible. By W. W. James, F.R.C.S. 29

The Treatment of Children from the Dental Aspect. By J. F. Colyer, M.R.C.S., L.D.S. 33

February 24, 1908.

The Dental Uses of Paraform. By J. E. Spiller, L.D.S. 41

On the Termination of the Nerves in the Teeth of Mammalia. By W. J. Law, L.D.S. 45

March 23, 1908.

Cases and Specimens:— PAGE

(1) Heads and Jaws of the Adult Hemirhamphus: (2) Developmental Defects occurring in the Upper Jaw of a Pike (*Esox lucius*). By A. HOPEWELL-SMITH, M.R.C.S., L.D.S. 61

A Case demonstrating a Point in the Treatment of Pyorrhœa. By ERNEST STURRIDGE, L.D.S. 62

Discussion on Mr. J. F. COLYER'S Paper, "The Treatment of Children from the Dental Aspect" 68

April 27, 1908.

A Contribution to the Study of the Movements of the Mandible. By NORMAN G. BENNETT, M.B., L.D.S. 79

A Case showing Incomplete Eruption of the Temporary Molars. By H. W. TREWBY, M.R.C.S., L.D.S. 99

May 25, 1908.

Notes on the Extraction of an Upper Wisdom Tooth. By W. RUSHTON, L.D.S. 101

Some Clinical Notes on Pyorrhœa Alveolaris. By J. G. TURNER, F.R.C.S. ... 104

Heredity and Dental Disease. By STANLEY P. MUMMERY, M.R.C.S., L.D.S. ... 108

June 22, 1908.

A Case of Diffuse Fibromata of the Gums. By ARTHUR EVANS, M.S. 125

Supernumerary Teeth. By R. McKAY, M.R.C.S., L.D.S. 129

A Difficult Extraction. By ARTHUR W. W. BAKER, M.D., L.D.S. 130

A Classification of Dento-Facial Irregularities. By J. SIM WALLACE, D.Sc., M.D., L.D.S. 140

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Odontological Section.

October 28, 1907.

Mr. J. Howard Mummery, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

Gentlemen,—My first duty, on taking the Chair at the opening meeting of our Section of the Royal Society of Medicine, is to thank you for the great honour you have done me in electing me your President for the second time, and especially at such an important epoch in our career.

It is a curious circumstance that our amalgamation with the Royal Society of Medicine should have taken place in the same year in which we have celebrated the fiftieth anniversary of the foundation of the Odontological Society. I think we can look back on fifty years of sound and useful work, years in which the speciality to which we owe our allegiance has made advances that the founders of the Society could scarcely have anticipated in that period, and I think that this union with the other medical Societies of London is a fitting climax to this fifty years of work and preparation.

The Co-operation of Scientific Societies which is now inaugurated is, I venture to think, a most excellent and wide-spirited movement. There has been in all departments of knowledge for many years past too great a tendency to specialisation and subdivision, too much exclusiveness, and in this excellent departure we look forward to a great advance in usefulness and believe that much benefit will accrue to all, in the interchange of thought and opinion that must accompany it. In our own Section we shall welcome criticism and no doubt profit largely by this transmigration, and we venture to think that the subjects under discussion in our meetings will bring those who practise other departments of the healing art more into touch with our speciality, and show them perhaps more clearly than they have hitherto appreciated how very intimately the health of the body generally is dependent upon the maintenance of a healthy condition of the mouth and teeth. As you all know, there have been great difficulties to be overcome in bringing together all these different Societies and in dealing with their various requisitions, and we owe a great debt of gratitude to the leaders in this movement for the persevering and patient way in which they have met all these difficulties and satisfied the most varied requirements of the different Societies, and have enabled us to start with a Society formed of thirteen sections representing every department of medicine and surgery. Those of us who have been on the Constitution Committee of the new Society are able better to appreciate the immense amount of work carried through by the honorary officers in the face of serious opposition and many difficulties. We hope that their efforts will be crowned with the full amount of success which they deserve, and that the Royal Society of Medicine has a long and successful career before it.

On November 10, 1856, the Odontological Society was formed, being constituted almost simultaneously with the presentation of a memorial to the Royal College of Surgeons for the institution of an examination by that body for a dental diploma. We know that at the time a great deal of feeling was manifested in the profession, and there was much criticism of the mode by which the constitution of the Odontological Society had been arrived at. Many considered, with some reason, that the scheme should have been submitted to a wider circle, and not have been left to the private decision of a few leading men in the profession, but by the light of later years we know that these leaders were far-seeing, and felt that the time had arrived when some great movement should be initiated for the elevation of the dental profession, and it also has to be remembered that in those early days when there was little professional feeling among dentists—when it was every one for himself, and the majority of those in practice feared what reforms might bring to them—an appeal to the whole profession might have resulted in signal failure. But the dismal prophecies as to the future of the Odontological Society in which many freely indulged were not fulfilled—the organised opposition of the College of Dentists at last gave way to wiser counsels.

The Odontological Society was soon firmly established, and, having gained an influence hardly to have been expected in so short a time, the amalgamation with the College of Dentists took place on May 4, 1863. Would that many of these brave men, who amidst all manner of discouragement and even abuse were staunch to their convictions, had lived to be present here to-night to see the result of their labours in the present position of the Society. The history of the Odontological Society is the history of the emancipation of our profession, and to the efforts of its members are due our recognition by the Royal College of Surgeons of England and the founding of the Dental Hospital of London and its Dental School. We are happy to-night to honour the men who fought so bravely for us in the past. Let us, like the Japanese, attribute all our victories to the virtues of our ancestors.

In those fifty years that have passed since the foundation of the Odontological Society what great changes have taken place, both in matters immediately connected with our speciality and in the wider field of general surgery and medicine! This period has seen a new science, Bacteriology, spring into being, which has thrown new light upon the diseases we are especially called upon to treat, and which in its very earliest days inspired Lord Lister (availing himself of the wonderful researches of the great Pasteur) to apply its teachings to the practical treatment of wounds. So universal is the adoption of this great reform in practice, that it is hard for us to realise what a boon to humanity this great discovery has proved. To those among us who, like myself, remember hospital practice in the sixties, it appeals with especial force. In my student days, however, I saw this great change take place. I saw suffering and death, supposed to be inevitable, proved to be preventable by this comparatively simple means, hospital gangrene and pyæmia banished, and compound fractures virtually turned to simple ones through the recognition by this master mind of the true cause of the infection of wounds. It may be interesting to recall the circumstances under which this great reform was brought about.

In 1860 a fine new infirmary was built at Glasgow, fulfilling in most respects the sanitary requirements of a great hospital; the ventilation and sanitary arrangements were considered excellent, but, as Lord Lister says in a lecture on the antiseptic treatment published in 1869, to the great disappointment of all concerned, this noble structure proved extremely unhealthy. Pyæmia, erysipelas, and hospital gangrene soon showed themselves, affecting especially the wards on the ground floor, which included the accident ward under Lord Lister's charge. In all cases of open wounds he found the proportion of fatal cases largely increased, especially when these cases exceeded a certain percentage; so bad was the condition on several occasions that the wards had to be closed for some time. The mortality becoming excessive, an investigation was made into the state of the building. "A few inches below the surface of the ground, behind the two lowest male accident wards, was found the uppermost tier of a multitude of coffins, which had been placed there at the time of the cholera epidemic of 1849, the corpses having undergone so little change in the interval that the clothes they had on at the time of their hurried burial were plainly distinguishable." "Yet at the very time when this shocking disclosure was made," says the author, "I was able to state that during the previous nine months, in which the antiseptic system had been in operation in my wards, not a single case of pyæmia, erysipelas, or hospital gangrene had occurred in them, and that, be it remembered, not only in the presence of conditions likely to be pernicious, but at a time when the unhealthiness of other parts of the same building were attracting the serious and active attention of the managers. Supposing it justifiable to institute an experiment on such a subject, it would be hardly possible to devise one more conclusive." The immunity from these diseases continued in the wards under his care during the remaining two-and-a-quarter years in which he remained in Glasgow, although it was also found later that at one end of the hospital the wards abutted upon a churchyard in which the pit burial of paupers was carried out, and around the infirmary, in pits each holding eighty, 5,000 bodies were lying in a state of decomposition.

I mention these facts to show what an absolute demonstration was given of the enormous value of this great discovery. In spite of this convincing proof, the new system, as is the case with every innovation, met with opposition in many quarters; and one of my earliest student recollections is that of a clinical lecture in which an eminent London surgeon declared that the new method of treatment of wounds had proved in his wards to be a failure. He was, however, soon afterwards convinced that the failure was due to imperfect methods, and became a warm advocate of antiseptic surgery. In our days it has become the only justifiable method of treating wounds, and its effects have been studied in all directions, in the treatment of all conditions of disease, with the greatest benefit to suffering humanity. Lord Lister's prophecy in 1869 has been amply fulfilled, when he said: "The antiseptic treatment is continually attracting more and more attention in various parts of the world, and whether in the form which it has now reached, or in some other and more perfect shape, its universal adoption can only be a question of time. The noble institutions of which our country is justly proud, admirably adapted alike for the treatment of the sick and the instruction of the student, will then be cleared of the only blot that now attaches to them." They *have* been cleansed from this blot, and it is impossible to estimate the number of valuable lives that have been saved since that time by the universal adoption of the antiseptic treatment.

In our own special department important discoveries and new methods have rapidly followed one another, one of the most important of these being the introduction of nitrous oxide gas as an anaesthetic, which we owe to Horace Wells, of Philadelphia, who, reviving the experiments of Sir Humphry Davy, made the employment of this agent a practical success. I remember the first administrations in England, when Dr.

Colton came from America and demonstrated its employment at the Dental Hospital in Soho Square. He used a tube held in the patient's mouth and brought direct from the gasometer. Even with this crude method of administration, although the blue colour of his patients from the impure gas employed was sufficiently alarming to the onlookers, he completely demonstrated the safety and efficiency of the new anæsthetic, which, with the valuable improvements in preparation and administration of recent years, has proved such a boon to us and to our patients. With this safe anæsthetic in our hands, it is astonishing that we should still see reported, from time to time, cases of death in the dental chair from the administration of chloroform. Why should we still have to emphasise the fact that it is not justifiable to administer chloroform to any patient in the sitting position? The introduction of the rubber dam and the dental engine, which we owe to our American cousins, has proved an immense boon to us in our daily practice, enabling operations to be more perfectly and rapidly carried out in the mouth.

More recently the introduction of pressure anæsthesia in the treatment of pulps has proved of great value, and local anæsthesia by injection bids fair to take the place of general anæsthesia in many small operations. During the lifetime of the Odontological Society we have lived to see miracles come to pass—the telephone, the phonograph, wireless telegraphy, the Röntgen rays; we do not consider them miracles now, but in former centuries they certainly would have been looked upon as such. Other discoveries quite as wonderful as these are yet in store for future generations, perhaps in the realm of physical science, perhaps in that of physiology. There are many mysteries to be fathomed, many problems to be solved; the mind of man has done wonders in the past and has capabilities still undeveloped. Let us say nothing is impossible; we have never reached the goal, but there are always fields still unexplored, countries still unnamed. Science has reached out her scaffolding into the dimness of the past, where she has hung out her lantern to illuminate the darkness still surrounding the dawn of life—she has, taking her stand on the firm foundation of established fact, thrust forward her piers into the dim vista of the future, and foreshadowed discoveries yet to be made by future generations.

So many worlds, so much to do,
So little done, such things to be.

I would like to dwell upon the fact that our Society represents the scientific side of our special branch. It was at its foundation designed to be a scientific society, and has established a fund for the endowment of scientific research. By calling it a scientific society, one does not for one moment attempt to convey that questions of practice and detail should be excluded, but that its papers and discussions should be devoted to both abstract scientific questions and to practice founded on a scientific basis; and we always find that the soundest practice, the most successful treatment, is founded on an accurate knowledge of facts. Empiricism, which may be defined as "an undue reliance on mere individual experience," has pitfalls for the unwary into which they frequently are plunged, and which they might have avoided had they taken their way along the firm roadway of knowledge to the sound practical results for which they had been striving.

Although much scientific work is undertaken and should be undertaken without any utilitarian object in view, there are many cases where such an object is in no sense derogatory to scientific research, and where most useful results may be obtained by investigations undertaken by scientific methods for the express purpose of obtaining some wished-for practical result. As an example of what may be done by a single man basing his practice upon scientific experiment and accurate knowledge, never empirical, never coming to any conclusion until arrived at by careful experiment, I may mention the late Professor Miller, whose recent loss we so deeply deplore. By the death of Professor Miller we have lost one of the noblest ornaments of our profession, one who, during his comparatively short career, has done an amount of work that makes us all feel pigmies beside him. He has earned the admiration and respect of the leading men of science in all countries, and the amount of scientific work he has done and the untiring devotion he gave to research can scarcely be appreciated except by those who knew him intimately. Beside his great work in clearing up the pathology of caries, there is not a department of dental science and practice in which he has not largely advanced our knowledge, and the members of this Society must vividly remember the absorbingly interesting way in which he dealt with many subjects at our meetings. His beautiful preparations are familiar to us all, and they alone represent an amount of work and of originality that is astonishing. Of his personality I am well able to speak, for I have lost in Professor Miller my best and most intimate friend. His personal charm was great; with all the enthusiasm of the man of science, he was a perfect boy in spirit, and took the keenest interest in all games and exercises, in which he invariably excelled. To myself, as well as to all who knew him, his loss is irreparable, and to the profession, both here and abroad, very difficult to estimate. Well may our American brethren be proud of such a man. The many Societies of which he was the life and soul will be as sheep without a shepherd, and it is very difficult to realise that his genial spirit has passed away from us. The name of Miller will live as a household word among us as long as our profession endures.

We have to-night taken a bold step; we have finally merged our individuality into that of a greater whole, but it is not a ceasing to be, not a termination of our existence, but a re-incarnation. We have passed from one state of being into another, but we have done so with all the advantages of the memory of our previous state, able to profit by it and improve upon it. The Greek philosopher Empedocles said: "For I have been in bygone times a youth, a maiden, and a flowering shrub, a bird, yea, a fish that swims in silence the deep sea." If re-incarnation is thus progressive, what may not lie before us? The sad part is, we cannot remember our former existences. Think, if one had been a reptile in early Pliocene times and could not now remember it, what opportunities we should have lost. We might have solved the problem of the dentition of the earliest mammal, we may have had a brief and painful opportunity of learning if he was truly triconodont, and settled many a debated point in palæontology, or even have been the missing link himself, and our previous bones still be hidden deep in some unexplored strata of the globe.

The Odontological Society has endeavoured to do its duty in the past; I think we can now hail with great satisfaction its incorporation with the new parent Society; and while wishing the greatest success to this new enterprise, and a glorious career for the Royal Society of Medicine, we can ourselves look forward with confidence to a wider influence and a greatly increased usefulness.

> "Who loves not Knowledge? who shall rail
> Against her beauty? May she mix
> With men and prosper! Who shall fix
> Her pillars? Let her work prevail."

The Teeth of Fossil Fishes.

By John Humphreys, M.D.S.(Birm.), L.D.S., F.L.S.

The teeth of fossil fishes are indestructible, and their structure can be almost as clearly demonstrated as those of fishes of the present day, although they were buried in the mud of primaeval oceans, in ages so remote as only to be reckoned by aeons of time. Since then, their organic constituents have entirely disappeared, and the teeth have been fossilised into rock as hard as the matrix in which they are embedded.

Among the higher Mammals dentine is of a simple character—hard or orthodentine, and a section of such a tooth discloses a central pulp-chamber, surrounded by its system of dentinal tubes; but as we descend the scale to lower types we shall see the structure considerably altered, and more complex in every way, from the greater vascularity of the tissue.

In Edentates we find the teeth of the sloths are formed of a dentine permeated by vascular canals, hence termed vaso-dentine. The megatherium, the gigantic extinct sloth of South America, exhibits this variety very clearly, in which the dentine and the cementum also were rich in looped blood-vessels which traversed the structures.

The teeth of the manatee possess similar characteristics, and in the singular ant-eater of the Cape, the orycteropus or aardvark, we see a still more complex form, in which the central pulp has broken up into a number of smaller branches, each giving off its system of dentinal tubes, thus producing a pattern bewildering in its complexity.

In Reptiles exactly the same condition of things is noticed. The snake and crocodile possess teeth of hard or unvascular dentine, but the further we go back in geological time the more complicated we shall find the dentine, and more richly vascular.

The simpler form of plicidentine is shown in a section of the tooth of ichthyosaurus, the outer surface being wrinkled by flutings; the blades of pulp, being squeezed laterally, are prolonged into the undulations, each blade giving off its system of dentinal tubes. If, however, we descend to the Carboniferous system, and examine the teeth of the archaic batrachians, the Labyrinthodonts, we shall discover, as the name denotes, the most singular and puzzling form of tooth substance known to us.

The central pulp-chamber, as in ichthyosaurus, gives off lateral arms or branches; but, instead of travelling straight to the periphery, each blade pursues a tortuous course, which adds greatly to the perplexity of the structure. The higher fishes illustrate this rule. Many of the osseous fish are furnished with teeth in which hard dentine, or osteodentine, plays an important part. Such are shown in the large conical teeth anchylosed to the sides of the lower jaw of the pike, upon which its prey is impaled when first seized, and by the strong teeth which arm the mouth of the angler fish. It is true that we have in the Cod family—as the hake, ling and cod—teeth which are eminently vascular, those of the hake being quite pink when alive, from the red blood circulating in loops throughout the calcified matrix. But in the more primitive group of Palæichthyes a complex form of structure still holds good.

The dawn of vertebrate life on this planet is being constantly pushed further and further back into the very remote periods of geological time. The scientific world was much exercised when Hugh Miller and Murchison published their discoveries half a century ago in reference to enormous varieties of fish-remains in strata as ancient as the Old Red Sandstone. The cutting of the Malvern and Hereford Railway disclosed the passage-beds tilted on end near Ledbury Station, rich in archaic fossil fishes; and the horizon was further carried into the Silurian system, to the fish-bed of the Upper Ludlow Series, only a few inches in thickness, but rich in remains of scales and teeth of small elasmobranch fishes, ancestors of the sharks and rays. Professor Lapworth tells us that the bed may be traced at intervals for more than a thousand miles, and only recently our knowledge has been enriched by Dr. Traquair's discoveries in Scotland in the Downtonian Beds, which there extend to several hundred feet in thickness, revealing the forms of the same small and singular creatures whose teeth and spines are agglomerated in the Ludlow Fish-beds, and which are, up to the present, the most ancient fish forms known. Their bodies were protected by enamelled spines and scales, constituting a shagreen similar to the covering of the modern dog-fish.

A study of the dental apparatus of the modern sharks and rays will enable us to understand the fossil teeth which abound in the Palæozoic and Mesozoic rocks. The jaws of the sharks are cartilaginous, with an outer slightly ossified crust. The teeth are extremely numerous, arranged in several rows one behind the other; they are not fixed in sockets or anchylosed, but united simply to the dense fibrous membrane which covers the jaws. One row of teeth stands upright; the reserve teeth lie in a recumbent fashion, protected by a flap of mucous membrane, and assume an erect position when the older teeth are lost. The dentition of the rays is similar; the teeth form a pavement similar to a Roman mosaic, and cover closely the dentigerous surface of the jaws. The modern rays are small and insignificant in comparison with their fellows of the Cretaceous period, such as the ptychodus, whose teeth I shall show you later on. Being produced in endless numbers and speedily lost by reason of their slight attachment, the teeth of sharks and rays are extensively found in marine geological strata, and in their attachment and mode of development they have been unaffected from an evolutionary point of view all through the stages of geological history.

The Old Red Sandstone is extremely rich in fantastically-formed fishes, suggesting almost a "'prentice hand" trial by Nature, so singular, grotesque and inelegant are the forms which have been brought to light, and which are arranged in a group—the Ostracodermi. Their bodies were protected either by bony plates, or an armour of scales, or enamelled scutes, which, fitting closely, formed an efficient protection from their enemies. Some, as the pterichthys, the coccosteus and bothriolepis, have the body enclosed in tightly-fitting plates, like the shell of a tortoise, and were propelled by oar-like appendages, and steered by the flexible tail as a rudder; others, as the cephalaspis, had the head enclosed in one great buckler plate, while the body was encircled by lateral plates. Pteraspis had the head surrounded with powerful bony plates, and the hinder part of the body by ganoid enamelled scales. These fishes, according to Professor Lapworth, "are usually regarded as primitive ganoids." They are cartilaginous in structure, like the sharks and rays, possessing no bony skeleton, and they were distinguished, like the ganoids which succeeded them, by tails which were either "diphycercal"—that is, the termination of the vertebral column was surrounded by the caudal fin—or "heterocercal," where the column is prolonged into the upper bifurcation of the tail, which is unequally divided, the lower lobe being the smaller. thus differing from osseous fish, where the tail is always "homocercal" or equally divided. The structure of the teeth was extremely beautiful, a transverse section of Dendrodus revealing a maze of pulp sinuosities comparable to branching trees, which suggested the name. the main stem and arms developing anastomosing dentinal systems of complex ramifications.

The Carboniferous Limestone contains numerous examples of the teeth of primitive sharks which abounded in the seas of that period. We possess in the *Cestracion Philippi*—the Port Jackson shark of the Australian seas—one of the few representatives of this once numerous family, whose dentition enables us to understand clearly the tooth forms of the Carboniferous rocks. The largest cestraciont sharks do not now exceed five feet in length, dwindled and insignificant indeed in comparison with the monsters which dominated the oceans of the distant past when our Coal Measures were being laid. The teeth of the cestracion are fixed to a dense fibrous gum, as in other sharks, and are lost and replaced by reserve rows, as in ordinary selachians; but the teeth in the front of the mouth are small and pointed, while the back of the jaw is furnished with rounded molariform teeth, an admirable adaptation for their peculiar diet, which consists of crustaceans and hard-shelled creatures, which are seized by the pointed teeth and crushed by the powerful molariform ones. Orodus, judging by the size of its teeth, was probably the most gigantic cestraciont shark which has ever lived.

The specimen before you measures four inches in length, and was found in the Cheshire Limestone; others have been frequently discovered in beds of the same age in Ireland, and also at Bristol. A powerful cone occupies the centre of the tooth, supported by transverse rugæ or wrinklings, which are separated by grooved furrows, their roughened surfaces rendering them peculiarly fitted for gripping and crushing shell-fish and crustaceans. The structure shows the well-known plicidentine character of these early fishes; long channels of pulp, running parallel to the surface of the crown, give off their systems of delicate tubes, which are specially evident in a transverse section, where the alternate pulp-chambers, ramifying tubes, and calcified matrix form a distinctive and beautiful pattern.

Sections of other Carboniferous cestracionts demonstrate the same structural features, as seen in the psammodus, helodus and psephodus. In the Coal Measures themselves we frequently find the bifurcated teeth of diplodus, which at first sight suggest a predatory shark, but they are evidently the teeth for prehension, of a cestraciont fish, similar to those in front of the jaws of the Port Jackson shark. In the later rocks of the Jurassic system we have excellent examples of the prevalence of this type of fishes, as in hybodus of the Lias, where the teeth are ridged somewhat like those of orodus, and the plicidentine is well marked, the outer border exhibiting richly tubular enamel.

Acrodos, from the Lias of Lyme Regis, shows a number of delicate groovings upon the masticating surface of the tooth, giving it the appearance of a fossil leech; while the strophodus bears a strong resemblance to the molariform pavement of *Cestracion Philippi* in the rounded plate-like teeth; both are excellent examples of richly vascular dentine and form very beautiful microscopic sections, showing the pulp canals, dentinal tubes and matrix, as clearly as in a modern tooth.

The Chimæras, a sub-class of the Elasmobranchii, made their first appearance in the Jurassic times, and differ remarkably from the sharks in their dental arrangement, as, instead of possessing detachable teeth, the jaws are furnished with dentigerous plates, which are never lost; four occur in the upper and two in the lower jaw. The true tooth character is made evident by a section, where, as in ischyodus from the Stonesfield Slate, and edaphodon from Eocene and Miocene rocks, we see the dental structure.

The ancestors of the Ray family are seen as low down as the Carboniferous Measures, but they attained their maximum development in Cretaceous times, the teeth of ptychodus indicating a fish of gigantic proportions. They are extremely beautiful in shape and structure, the surface of each tooth being roughened by sharp ridges and sinuous grooves, the circumference covered with minute rounded cusps to enable the creature to effectually grip and clutch its prey. A horizontal section shows the pulp canals more numerous, and dentinal tubes finer, than in earlier specimens.

In early Tertiary measures we find the first appearance of the singular rays, myliobates and aetobates, where Nature has substituted dental plates for teeth. In the modern myliobates both jaws are covered with such plates, forming a dentigerous pavement, admirably suited for crushing shell-fish; the plates fit closely together, each consisting of comb-like denticles on the under surface, which become fused into a dense, smooth, polished upper surface, attached to a fibrous gum only. Myliobates was very widely distributed, fossil specimens being found in Eocene and Miocene measures in many parts of the world.

So far we have been discussing the varieties of dentitions of fossil cestraciont sharks and rays, and now we have to return to Carboniferous Measures to denote the advent of higher forms of fish which bear a certain resemblance at first sight to osseous fish of recent times. We noticed in the Old Red Sandstone the archaic forms and armoured bodies of the group Ostracodermi; these, in the Coal period, were followed by ganoid fishes, whose bodies were enclosed in accurately-fitting enamelled scales, but possessed no ossified skeleton. We have a few illustrations still surviving of this rapidly disappearing class, which figured so largely in Mesozoic times, in the sturgeon and lepidosteus, or bony pike, of North America. Unlike the Palæichthyes, the teeth were anchylosed to bony jaws, and indicate by their shape their predatory character.

We have illustrations in rhizodus of the immense size of its jaws and teeth, which probably represent the most gigantic fishes, apart from the sharks, which have ever inhabited the seas; a comparison with the jaws and teeth of the modern lepidosteus suggesting the difference in their dimensions. The teeth are conical, compressed laterally, the anterior and posterior surfaces developed into sharp ridges with a knife-like edge, the lower part grooved, indicating a tooth of powerful trenchant character. My specimens measure $3\frac{1}{2}$ inches in length and $1\frac{3}{4}$ inches in breadth, but they are small in comparison with some in the Royal Society of Edinburgh, where a fragment of the jaw, $18\frac{1}{2}$ inches long, is furnished with teeth measuring 5 inches from base to point, and the scales which protected its body were 5 inches in length. Well might

Agassiz describe it as "the largest of osseous fishes"! In one of my specimens you will notice two enormous teeth standing side by side, which armed the front of the lower jaw as teeth of prehension, reminding one of the canine tusks of the Carnivora, and we can well imagine that rhizodus was the terror of the Carboniferous seas. In no other geological period have we evidence of such extensive and varied fish life, and it is singular that the Cestraciont sharks and the ganoid fishes attained in those measures their maximum development at a period so early in the world's history.

Megalichthys was a companion fish of huge dimensions, as its name implies. The teeth were more attenuated than in rhizodus, but similarly fluted at the base; they are found in the Carboniferous Limestone and the Coal Measures proper. The open coal workings at Tipton have furnished great numbers of its teeth, the structure of which, as in rhizodus, was a rich plicidentine.

Through the various stages of the Jurassic deposits we find interesting varieties of ganoid fish and their dental apparatus, of which we may take lepidotus as a type. All the specimens which I am about to show you had the same habits, their diet consisting of shellfishes and crustaceans, for crushing which the powerful teeth which covered their palates were well adapted. In lepidotus they were rounded and berry-like, with a point occasionally upon the upper surface, hollow inside. The teeth of sphærodus, from the Greensand, are similar, having the appearance, when inverted, of acorn cups; the successional teeth, which are numerous, when erupting, turn over at an angle of 180°. Pycnodus, phyllodus, pisodus, mesodon and microdon exhibit admirably the arrangement of the teeth, which were anchylosed to the bones, varying in shape and size, but all admirably fitted for the work they had to perform.

The palatal teeth of gyrodus, from the Kimmeridge Clay, are very beautiful examples of crushing teeth, each being furrowed with a deep groove, to roughen the surface and increase the gripping power. In structure the Jurassic ganoids exhibit a decided advance upon their Carboniferous predecessors, a hard dentine with ramifying tubes, developed from one central pulp-chamber, having taken the place of the vascular teeth of earlier fishes. The enamel is very puzzling, the inner third, as in sphærodus and lepidotus, being penetrated by tubes which cross one another in an aimless fashion.

I must not omit to say that in Triassic measures we have the first appearance of the dipnoi or mud-fishes, whose representatives still exist in the ceratodus of the Queensland rivers and the lepidosiren of Western

Africa. The mud-fishes are furnished with lungs and gills, to enable them to live either in the water, or, when the rivers run low and dry up in the summer months, to exist in the hardened mud by means of their lungs. The curious dental plates which have been found so frequently in the Triassic and Rhætic beds have long puzzled anatomists, but their true character was demonstrated by the discovery of ceratodus in Queensland, about the year 1870, when its singular dental apparatus afforded a clue to the earlier fossil forms.

In both the upper and lower jaws of ceratodus the bones themselves are serrated and glazed with a coarse enamel, the plates in the lower jaw articulating with those of the upper. That the bone itself comes into play for dental purposes is a very extraordinary occurrence, and it is phenomenal to find the tissues strengthened and protected by enamelling.

The predatory sharks came into existence in Upper Cretaceous measures, of which we have illustrations in *Otodus obliquus*, *Corax falcatus* and *Odontaspis elegans*, while the Red Crag furnishes us with the triple-cusped tooth of notidanus, and the simple but powerful tooth of oxyrhina. They all present the same appearance of a plicidentine character.

In *Otodus appendiculatus*, from the Cambridge Greensand, the channels of pulp are extremely numerous, and the dentinal systems naturally more contracted and stunted; but the enamel is penetrated by a system of tubes, very wide, with large open mouths where they enter from the dentine, branching into meshwork for a third of their length, and then tapering finely towards the periphery of the enamel.

Odontaspis elegans, from Upper Cretaceous measures, shows a tooth of similar structure. The channels of pulp towards the surface are long and attenuated, and the enamel is penetrated with tubes from the *outer* surface.

Oxyrhina, a Miocene shark, shows a very beautiful vascular structure in the dentine. The fine tubes cross one another in a perfect maze, and the enamel is penetrated by a double system of tubes, from within, on the dental surface, where they branch into fine rootlets when they have traversed a third of the enamel; and from without, where they run in straight tubes from the surface of the enamel. Just beneath the enamel is a line of interglobular spaces, bearing some resemblance to the granular layer in human dentine.

The predatory sharks attained their greatest development in Miocene times, when the carcharodon dominated the Tertiary oceans, a creature, judging by the size of its teeth, which must have equalled a whale in proportions. Some of my specimens measure five inches in length by four in breadth, and are found in all parts of the world—in New Zealand,

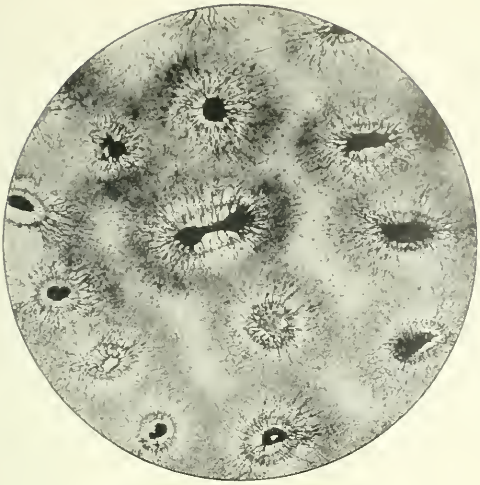

Fig. 1.
Ovodus.

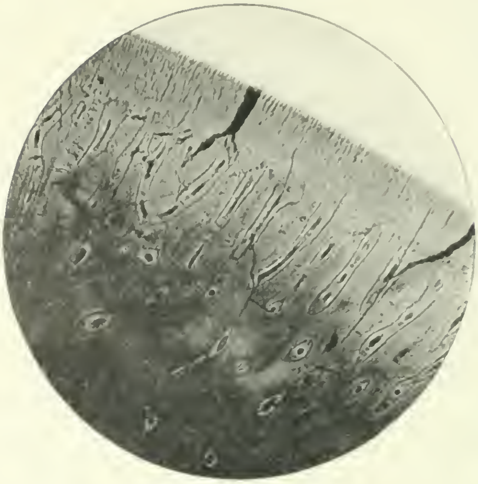

Fig. 2.
Odontaspis.

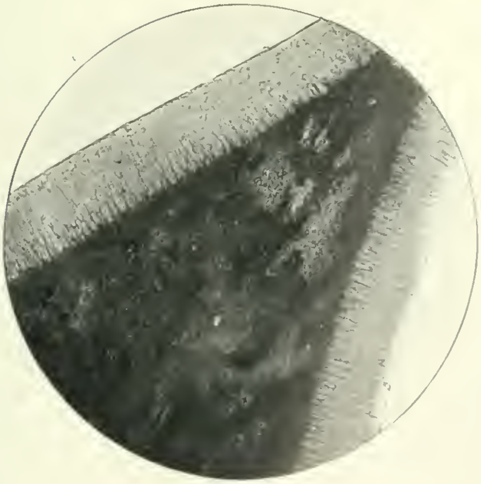

Fig. 3.
Otodus.

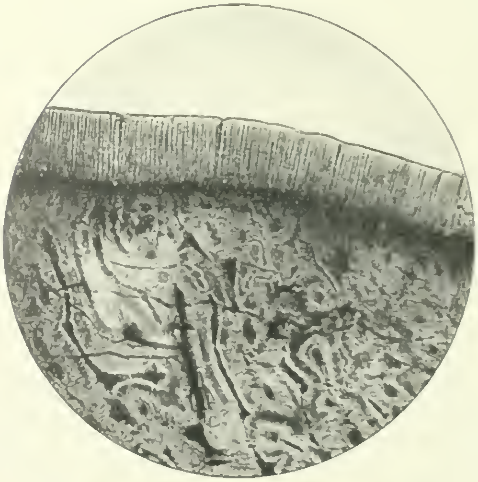

Fig. 4.
Carcharodon.

HUMPHREYS: Teeth of Fossil Fishes.

Australia, South Carolina, the Bermudas, and the Continent of Europe, besides our own Suffolk Red Crag, showing the extent of its world-wide distribution. Its teeth present an instructive illustration of tubular enamel; the tubes, entering from the dentine by wide open mouths, run in straight lines to the enamel surface, and they are continued right across, in a similar fashion to the tubes in marsupial enamel. The dental structure of modern sharks is one of osteo-dentine, graduating into fine tubed dentine on the surface of the tooth.

The osseous fishes, with truly ossified skeletons, like the fishes of the present day, made their appearance in Cretaceous times, but the specimens I possess are too few to describe their structure.

A few conclusions may be arrived at from the study of Fossil Fishes:

(1) The very early appearance of fishes in geological time, in ages as remote as the Upper Silurian measures.

(2) The apparently sudden appearance of cestracionts and ganoid fishes of gigantic size, which attained their maximum development in the Carboniferous seas.

(3) The rays, though contemporary with cestracionts in Carboniferous time, did not attain their maximum proportions until the Cretaceous period.

(4) Predatory sharks appeared first in the Upper Chalk, attaining their greatest size in the Tertiary.

(5) The remarkable continuity of species, as evidenced by the persistence of cestracionts, from the Carboniferous measures, to the modern Port Jackson shark of the Australian seas, and of ceratodus, from the Trias, to the present time.

(6) The earlier forms of fishes show a dental structure richly vascular, or plicidentine. The more archaic the form and remote the period, the greater the complexity of the tissue.

(7) The extraordinary variety of arrangements provided by Nature in fishes for the prehension and comminution of food, as illustrated by the molariform hinder teeth in the jaws of the Port Jackson shark; by the dental pavements of myliobates and aetobates, the dentigerous jaws of edaphodon, the various and beautiful palatal teeth for crushing shell-fish, as seen in gyrodus, sphaerodus, lepidotus and ptychodus; and, lastly, by the enamelled bony plates of ceratodus.

DISCUSSION.

Mr. D. P. Gabell said he was very much struck by the enormous size of the enamel tubes shown in some of the specimens. The current explanation of ameloblast cells scarcely covered those very large tubes, and he would like to hear whether there was any other theory to account for them.

Mr. F. J. Bennett said that while congratulating the reader of the paper on the splendid collection of slides, and on the way in which he had set forth the subject, he wished to ask whether the matter might not have been pushed a little further in one or two directions. Mr. Humphreys' opening statement was that all those structures represented the disappearance of soft tissues, and their replacement by inorganic deposits. Recently that had been somewhat questioned with regard to some fossil forms. At the Natural History Museum the authorities had succeeded in decalcifying some of the specimens, and finding beneath some remains of more resistant organic material. He asked whether Mr. Humphreys had tried, and if not would he be disposed to try, the same thing on some of the fishes, to recover some of the original dentinal tubes. Possibly some organic structures might be found which were quite hidden by the calcified deposits. Another enquiry might be opened up in connection with the chemical nature of the deposits found. One knew that iron entered largely into the new structure, and if Mr. Humphreys felt that his energies were equal to the task, further research along those lines would be very interesting. He concluded by congratulating the author on his admirable paper.

Mr. H. Baldwin said it might perhaps be looked upon with a degree of pride by dentists that the teeth of the creatures exhibited formed such a large part, morphologically, of the animal; so much so that in many of the cases the animal derived its name entirely from the tooth. Possibly in some cases the tooth was the only part of the animal discovered.

Mr. Humphreys, in reply, thanked the members for the very kind reception they had given to his paper. He felt it to be a very great honour to be asked to deliver the first address before the new Section. In answer to Mr. Gabell, it was very difficult to say what was the use of the tubes in enamel. Mr. Tomes had written, in the *Transactions of the Royal Society*, a paper, which no doubt Mr. Gabell had read, on the structure of the enamel in elasmobranch fishes, and he brought out very plainly its tubular character and structure. Perhaps it was not necessary to make those structures so hard in teeth which were used only for a short time and then replaced by others. He did not know whether it was so, but be could not offer any real explanation. It was found to persist to the present day. Mr. Bennett had suggested that some of the specimens could be decalcified. He (Mr. Humphreys) promised to act on the suggestion, though he had not much hope of finding anything organic after the lapse of so many millions of years, during which they had been fossilised into rock. Mr. Baldwin's remark with regard to the important part played by the teeth in life impressed him greatly, because one felt how much the teeth contributed to the story of the evolution of life, from the beginning of geological time. As he remarked in the paper, he looked upon the specimen showing scales and teeth, from the Upper Ludlow beds of the Silurian system, with awe and amazement, when realising that there was the dawn of vertebrate life. During those geologic ages one found that everything had disappeared except the teeth, and it remained for the teeth to tell most eloquently the story through such immense time.

Odontological Section.

November 25, 1907.

Mr. J. Howard Mummery, President of the Section, in the Chair.

Some Notes on the Growth of the Jaws.

By James T. Carter, L.D.S.

I have chosen the subject of "The Growth of the Jaws" knowing full well that there are many members of this Society far more competent than I am to deal with it. But there are some aspects of the case which I think have been insufficiently commented upon. The consideration of the development and growth of the jaws, and of the variations and of the correlations which exist between the jaws and other facial and cranial bones, forms a subject of great interest to all dentists. Fortunately for us as a profession, the consideration of this same subject is also of great importance to anatomists, anthropologists, and others, so that the problems surrounding it are being investigated by a large and growing body of workers. To deal with the whole question of jaw growths one must consider:—

(1) The development of the maxilla, premaxilla, and mandible, also the normal changes which take place in these bones after birth.

(2) The relation of the jaws to other facial and cranial bones, together with osseous and muscular variations.

Then, with some definite knowledge concerning the elements of the face and cranium, we are able to turn to (3) the examination of large groups of individuals, and to endeavour to establish correlations. And, lastly, there is (4) the physiological aspect of growth. This forms far too great a field for me to attempt to deal with, but I hope the few notes I shall give this evening will be of interest.

The first point to which I would draw attention is to the development of mandible, maxilla and premaxilla. Professor Edward Fawcett, who has done a great deal of work on the development of cranial and facial bones, has kindly furnished me with particulars of his investigations into the development of the maxilla and of the mandible, and though many of his conclusions and results are as yet unpublished, he has given me permission to make reference to them. His researches were carried out by means of serial sections, and the use of the reconstruction method. Thus he was enabled to eliminate the sources of error which misled earlier observers.

In the mandible Meckel's cartilage is somewhat late in its appearance, not becoming cartilaginous until after the femur, though its *anlage* is visible in the 12 mm. to 13 mm. embryo. When formed the two cartilages, seen in horizontal sections, form a horseshoe, incomplete at first, being separated by an appreciable interval. Soon, however, they meet in the middle line, and later usually overlap one another there. Each cartilage is thickened in the vertical sense on each side of the future symphysis. If we trace the cartilages backwards from this anterior thickening, we find that they form a large bend with the convexity outward, and still further backward form another bend with the concavity outward, before ending in the malleus. The latter bend is found in front of the Eustachian tube. In its concavity lies the inferior dental nerve, and against its convexity the lingual nerve.

Ossification commences in front of the anterior expanded end of Meckel's cartilage, and the incisive branch of the inferior dental nerve, and extends backward under the mental nerve as a strip of bone rapidly diminishing in thickness. At this time there is no sign of development of an inner alveolar border. The inner alveolar border arises by ingrowth from the main mass just behind the mental nerve, in the form of an obliquely directed shelf, coming to a point behind the lingula, and this shelf passes between the inferior dental nerve and Meckel's cartilage, which it overhangs. At no time is it completely separate from the main mass. In other words, there is no splenial centre. At first there is no inner alveolar border behind the incisive nerve, but a shelf grows backwards over Meckel's and under the incisive nerve, near its origin from the inferior dental nerve. Thus the inner alveolar border arises from the main mass in two ingrowths, the first under the inferior dental nerve, enlarging in the backward direction and terminating in the "lingula," the other growing under the outer end of the incisive branch of the inferior dental nerve and extending inwards towards the middle line. Ossification begins in Meckel's cartilage about the tenth week in the region of the mental foramen and gradually extends inward, so that the portion of the jaw between the mental foramen and the symphysis is partly cartilaginous, partly membranous. This is the only portion of Meckel's which undergoes ossification, for posterior to the mental foramen the cartilage undergoes atrophy. The canal for the nerves is completed by the growth of spicules from one alveolar border to the other over the top of the nerves, the mental nerve being first so covered, then the incisive, and much later the inferior dental near the permanent inferior dental foramen. An accessory mass of cartilage, quite independent of Meckel's, appears to form the condyle proximally, whilst it tapers away distally to a point terminating in the root of the coronoid process. This plug of cartilage is enveloped everywhere, except at the condyle, by membrane bone, and is ossified itself very early. Another patch of cartilage appears along the anterior border of the coronoid process, also quite independent of Meckel's and the condylar wedge. Kollman has shown that other patches of cartilage occur at the angle and also along the outer alveolar border in the neighbourhood of the incisor and canine teeth, but according to Professor Fawcett's observations, this is not ossified independently (this patch on the incisor region is of interest in view of the opinion of Professor von Bardeleben, that the chin in man and other mammals is a distinct element). In all probability these cartilaginous plugs do not represent separate morphological elements, but are adaptations to the growth of the jaw. Thus we find that the lower jaw is developed in membrane as a single element, and the old view, which held that there were a number of separate centres of ossification, homologous with the elements of the reptilian jaw, is incorrect.

The maxilla arises as a membrane bone exterior to the cartilaginous nasal capsule, and in the neighbourhood of the canine tooth germ, by one centre of ossification. Its first appearance is in the fœtus of about 18 mm. in length, and it is the third bone to appear, the clavicle being first and the mandible second. From this centre three processes rapidly radiate out—

(1) A nasal process which grows upwards.

(2) An alveolar process which grows downwards, is thickened at its root to form the malar process, and might be called the alveolo-malar process.

(3) A palatine process which grows inwards.

The alveolo-malar process grows backwards, fork-like, and between the two limbs of the fork the anterior dental nerve grows in order to pass forwards under the alveolo-palatine angle of junction. These parts are all well developed at 19 mm. At about 32 mm. cartilage is developed in the outer alveolar border in the neighbourhood of the milk molars, and this cartilage is ossified by extension of bone from the main mass. Soon after this the internal alveolar border appears as a down-growth from the palatine process. At this stage the bone is very flat behind the nasal process and the eyeball is only separated from the tooth-germ by a thin plate of bone. At first the infraorbital nerve lies on the top of the orbital surface, but, later, ridges of bone grow up on each side of it ultimately meeting over the nerve and forming the infraorbital tunnel. Frequently a suture remains here which has been erroneously taken by Sutton, Polot, and others to indicate double ossification.

The antrum of Highmore appears as an outgrowth from the cartilaginous nasal capsule and separates the orbital floor from the roof of the tooth crypts; consequently this raises the total height of the maxilla and alters its form. The maxillary antrum at first is lined by cartilage, but later this undergoes atrophy and a mucous lining alone remains.

With regard to the ossification of the premaxilla, Professor Fawcett found great difficulty owing to its very irregular appearance, which is usually just after the maxilla, *i.e.*, about 19 mm., in the form of small scattered masses of bone especially congregated over the two incisor teeth and forming a sort of bridge over the interval between these teeth. From that portion in the neighbourhood of the lateral incisors a part of the outer alveolar border is early formed, likewise a part of the nasal process. Union between the premaxilla and maxilla commences soon afterwards, at 22 to 24 mm., at the alveolar border and extends upwards into the nasal process. At a later stage, about 50 mm., the premaxilla consists of two parts: (1) a processus lateralis or facial part, and (2) a processus medialis, which runs backwards on the mesial side of Stenson's canal. This mesial part—the so-called centre of Rambaud and Renault—is not a separate centre of ossification, at any rate normally, but is simply a backward growth of the premaxilla, and its mesoblastic *anlage* is easily to be seen growing backwards and upwards from the premaxilla. In many quadrupeds it is ossified independently. At 42 mm. there grow backwards from the facial portion of the premaxilla, two wedge-like processes to meet the palatine processes of the maxilla proper which cause the two sutures one sees on the under surface of the whole bone at birth and later. So it will be seen that the maxilla develops from one centre only, and that the premaxilla also has but one centre. At first there is no anterior alveolar wall to the two incisor teeth, and the anterior dental nerve may be seen sweeping round to the front just over these teeth; later, a downgrowth of bone takes place and hides teeth and nerve from view.

In a very interesting paper on "The Expansion of the Maxillary Sinus," Dr. Arthur Keith has shown that the growth of the maxilla and the development of the antrum are closely related, and that the growth and changes in the lower jaw are correlated with, but secondary to this development. He has also pointed out that all the permanent molar teeth are developed in the posterior border of the jaw in front of the pterygo-maxillary fissure and that owing to the expansion of the antrum this posterior border is carried downwards and forwards until it becomes the alveolar border. The effects of this growth are best seen in the gorilla where the expansion of the sinus is enormous. "That part of the jaw which carries the milk teeth and which at birth lies beneath the floor of the orbit is thrust downwards and forwards until it lies entirely in front of the orbit. Along with this growth downwards and forwards of the premaxillary part of the jaw, there is also a rotatory movement in the molar-carrying part of the maxilla, whereby the posterior border of the jaw moves downwards, its lowest portion becoming the alveolar margin. This rotation is best seen in the orang, in which the three molar teeth are permanently implanted on a segment of the circle in which the rotation takes place. The growth of the upper jaw in each direction is most rapid during the eruption of the permanent teeth. The alveolus occupies practically the whole depth of the posterior margin of the jaw at birth, in man and ape alike. While the posterior border of the jaw quadruples its extent from birth to maturity, the facial border becomes little more than double, and the alveolar margin increases in even smaller degree. Growth adds to the depth of the posterior part of the jaw much more than to any other dimension, and this addition is entirely due to the growth of the maxillary sinus." The fulcrum on which this rotation takes place is formed by the body of the sphenoid, the anterior surface of its great wing and the pterygoid plates and mainly by the internal pterygoid plate. Fawcett has shown that whilst the external plate is ossified entirely in membrane the internal plate is ossified in cartilage and that ossification begins here much earlier than has been supposed, having started on the 19 mm. fœtus. The cartilaginous origin of the internal pterygoid plate is very suggestive. Of course the maxilla does not come into actual contact with the sphenoid, the palate bone acting as a wedge between. It is interesting to note that ossification starts in the vertical plate of the palate bone immediately internal to the palatine nerves, about 19 mm., and the palatine process appears in the 24 mm. embryo. The dependence of the pterygoid plates on the development of the maxilla was pointed out by Sir John Tomes who showed that these increased between the ages of seven and twenty-one years by one third of their ultimate length.

In the chimpanzee there is great increase in the size of the intermaxillary bone between infancy and adult life as was pointed out by Mr. Charles S. Tomes. Whilst sectionising a number of skulls of apes I happened to bisect the skull of an adult chimpanzee and found a condition which I do not think has been described hitherto, *i.e.*, the existence of an intermaxillary antrum formed by a prolongation or off-shoot of the maxillary antrum. So far as I can see this starts on the antero-internal wall at some distance above the level of the floor, from thence it passes inwards into the palatal process of the superior maxilla, and then behind the enormous socket of the canine into the intermaxillary bone, its growth progressing until a deep depression is formed immediately behind the sockets of the two incisors. Another off-shoot starts high up on the anterior surface and passes over the top of the canine socket to join the cavity in the intermaxillary bone; this probably has some connection with the full eruption of the canine, for in specimens where the eruption of the tooth is not complete the junction of the two cavities has not quite taken place.

In certain monkeys and apes, sometimes in man, the maxillary sinus is comparatively feebly developed, and in these cases there is a great development of the inferior meatus of the nose, which brings about a result similar to the expansion of the antrum. In certain skulls of gorilla which I have there is no prolongation of the antrum into the premaxilla, but this is compensated for by a deep depression from the inferior meatus lying immediately behind the sockets of the incisor teeth.

With the inflection of the spheno-ethmoidal angle and consequent reduction of the forward projection of the maxilla there has been a shortening of the hard palate antero-posteriorly. This reduction of the bone is out of proportion to the reduction in size of the alveolar arch, for the teeth and alveolus involute more slowly. To meet this reduction the teeth assume an arch of a different shape, and the palatine processes of the superior maxilla are greatly reduced. In white races the horizontal plates of the palate bones form a much larger proportion of the hard palate than in the case of negroid races, and this disparity seems to be still greater in the Australasian races. In anthropoid apes the transverse palatine suture is often in a line with the posterior borders of the wisdom teeth, and forms a very small part of the hard palate. Knowing as we do that the teeth are the organs which undergo least change in the phylogeny of the race, and that the alveolar arches are dependent for their existence on the teeth, this points to the fact that the changes in the maxilla are most marked in those parts which have no direct association with the teeth. I shall return to this subject again in dealing with the lower jaw.

The growth of the mandible is determined to a large extent by the growth of the upper jaw. At birth its upper border is almost on a level with the condyle. With the growth of the maxilla, consequent on expansion of the antrum, the mandible gradually assumes its adult shape, the angle being the last portion to appear. The width of the ascending ramus is largely dependent on the length of the mandible, and this again is correlated with the cephalic length, but with this exception the growth and shape of the jaw seems to be largely independent of that of the calvaria.

Professor Arthur Thomson has investigated influence of jaw growth on cranial form, and proves that there is a distinct correlation between head length and mandibular length; also that the longer jaws have, as a rule, the higher coronoid indices, *i.e.*, that in the case of a mandible with a high index the force is applied at a point further removed from the fulcrum than in a mandible with a low index, his mandibular index being

$$\frac{\text{Condylo-symphysial length} \times 100}{\text{Inter-condylic width}}$$

His investigations also show that dolichocephals are, as a rule, furnished with much more powerful temporal muscles than are brachycephals; also that the shorter mandibles of the brachycephals required a lesser force to produce the same result as compared with that which necessitated the greater force on the longer jaws. Taking the condyle as a fulcrum, the greater distance at which the force is applied the less the amount of muscular effort necessary; in other words, the broader the ascending ramus in proportion to the total length of the mandible, the less the effort needed.

There is another point in connection with the growth of the mandible which I think should be emphasised, and that is, that the teeth and tooth-bearing parts change least in the phylogeny of a race, whilst those parts of the bone to which the muscles are attached undergo the greatest change. With the progress of civilisation there has been a considerable reduction of the mandible quite out of proportion to the reduction in the size of the teeth. Allowing for variation in length of the jaw, owing to the correlation between head-length and jaw-length, I have found the greatest variation in the width of the ascending ramus. We find that the alveolar border is larger in proportion to the rest of the mandible in women than in men, and in civilised than in lower races.

In negroes and in Australian aboriginals there is generally a well marked space between the posterior border of the third molar and the anterior surface of the ascending ramus. Pelletier has measured this by means of Broca's stereograph in a large number of cases, and found that this averages as much as 4 mm.

In Europeans, however, she found that the ascending ramus overlaps the wisdom tooth by as much as 4 mm. From this fact she drew the conclusion that there is an unequal rate of growth of different parts of the jaw. Knowing as we do that the mandible is formed as a single skeletal element, which grows in length by deposition of bone on the posterior border of the ascending ramus and absorption from the anterior surface, we see that it is a case of arrested development.

DISCUSSION.

Mr. F. J. BENNETT said that the very interesting paper which had been read by Mr. Carter deserved members' thanks. A great deal of the matter which was brought forward in that paper seemed to him to be new, and in some respects differed from the paper which Mr. Bland Sutton read before them some years ago. Many of the points would require cereful reading and comparison to appreciate them, but when that was done, he felt sure much of value would have been learned. With regard to the point as to the discrepancy between the development of the jaw and the development of that portion of it which bore the teeth, he thought a reasonable explanation was the following. The teeth, in consequence of their slow development, had to be formed at a much earlier age than that at which they were needed, and therefore they would be considerably in advance of the growth of the jaw. It was observed in allied species at a very young age, indeed, that they resembled each other much more than in the adult condition. Therefore, one might consider that when the teeth were laid down, it was in anticipation of the jaw being developed to the full extent; whereas, by the time the jaw had become fully developed, the species had, to some extent, become modified, and taken on a new shape. Therefore, in the jaws, as completed, there were two stages of development; one which belonged to the early condition of the species and bore the teeth, and that part of the jaw which had become modified by more recent development, and which was, therefore, much smaller. There was a point concerning the development of the antrum which he did not know whether others had noticed. Taking the maxilla at the time when the temporary teeth were complete, it would be found that the first permanent molar lay in a crypt at the back, in a posterior and outward direction to the jaw, and on a higher level. If one followed out the growth of jaws at about that age, one would find that the teeth had become more or less erupted, and that what had been the crypt was occupied by the antrum. And so also with regard to the second and third molar teeth. One could see the marks of those crypts in the septa on the floor of the antrum, marking the position where the original crypt for the permanent teeth had been. The matter was worth looking into by anyone who was interested in the subject.

Mr. N. G. BENNETT said that Mr. Carter had made the very definite statement that in the progress of civilisation the mandible had decreased in size at a greater rate than the teeth themselves. Doubtless that was true, even to such an extent as to make it certain that the decrease was due to definite inherited characteristics, and not merely to the environment and surroundings of the individual concerned. If that were so, it seemed to bear very markedly on the different schools of thought existing at the present time with regard to the regulation of misplaced teeth, narrow arches, and crowded mouths, because it showed, if true, that children existed whose teeth were too large for their mouths. Therefore, the view which was held largely on the other side of the water, but also to a considerable extent on this side, that, with very rare exceptions, indeed—in the case of obvious deformities—every individual was born with teeth which were the right size for that individual, would be untenable, and the extraction of teeth to relieve crowding in certain cases—of course associated with treatment directed to the expansion of the jaw—would be quite justifiable.

Mr. H. BALDWIN said the present was scarcely the occasion for entering into a discussion on the question of regulating contracted arches; but in regard to the ætiology of contracted arches he wished to say that he thought the view held that the maldevelopment or arrest of development of the bones was due to difficulty in breathing through the nose, was correct, and that therefore it was a sort of accidental circumstance. Had it not been for that difficulty, the bones would have been sufficiently formed for the teeth to have assumed a regular position. On the other hand, there were, no doubt, certain cases, though they were rare, in which that disproportion between jaws and teeth was due to something else, and, as Mr. Bennett suggested, it would require to be treated by extraction. He thought that in the majority of cases the malformation of the bones was due to an accidental circumstance, namely, the difficulty of breathing through the nose. Therefore, the correct method of treatment in those cases was to try to put the teeth and jaws into their correct size and shape; and, possibly, if that did not succeed, to resort to extraction.

Mr. J. F. COLYER thanked Mr. Carter for his excellent paper. He thought contributions of that nature would go far towards solving some of the vexed questions which arise in practice. The point he brought forward as to the antrum causing irruption of molar teeth was one which had occurred to him, Mr. Colyer; and he had often wondered how far nasal insufficiency, by causing lack of function of the antrum, influenced the growth of the maxilla. As the author probably knew more about the growth of the jaws than most of the members, he wished to ask whether he could explain those curious cases of mandibular third molars which tilted forward. A misplaced molar very often did not occur in a crowded mouth. There might be many stages of that, from slight tilting to complete inversion. There were cases in which the condition was carried further, the third molar erupting in the sigmoid notch of the mandible. Also, did Mr. Carter think that those curious cases of teeth in the temporal bone of the horse could be explained by the tooth band being carried too far back? That seemed a likely explanation of some of the misplaced molars, and he wondered whether it accorded with anatomical knowledge.

Mr. CARTER, in reply, said the point he wished to emphasise in his paper was, that before one could arrive at a satisfactory knowledge on the growth of the jaws, the whole question had to be considered from its beginnings: First of all, the development and ossification of the various bones which were related to the jaw, followed by the study of their normal natural growth after birth to adult age. Until that was carefully worked out, there would never be any real definite basis on which to build a knowledge of the cause of irregularities. But in regard to the causation of irregularities of the jaws and of the position of the upper jaw, one bone had to be carefully studied, namely, the sphenoid. The inflection of the angle between the sphenoid and the ethmoid was much greater in some cases than in others; and in the crossings of races, or of different types, it was very marked. A slight growth of the maxilla, with the pterygoid plates acting as a fulcrum, would push the jaw much further forward; and he thought that was often the cause of the change of position of the third molar to which Mr. Colyer alluded.

The Effects of Chronic Suppuration in the Molar of a Horse.[1]

By J. G. TURNER, F.R.C.S.

The tooth showed towards its basal part four sinuses in the dentine in the smooth everted edges, communicating with the pulp cavity; about half-inch more of the tooth had been formed since the date of origin of the sinuses. These latter were evidently formed in soft tissue and calcified after. There had been toothache early in life, and a piece of alveolus had been cut away, giving exit to pus. The tooth had remained tender till death, the animal shying causelessly on a hard road. The pulp had been exposed on the coronal surface by softening (? caries) of the dentine, probably due to lodgment of oats, and had perished to its base where the more free collateral circulation at the widely open apex of a forming tooth allowed of a better resistance to infection. The case was comparable to the abscess-cavities found in the tusks of elephants.

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[1] Read by Mr. Watson Turner, in the absence, through indisposition, of the Author.

Odontological Section.

January 27, 1908.

Mr. HOWARD MUMMERY, President of the Section, in the Chair.

Specimens added to the Museum.

By J. F. COLYER.

THE HON. CURATOR exhibited some of the recent specimens added to the Museum. At the December meeting the Council had authorised the purchase of eighty or ninety skulls, which had been placed in the museum, and a few of which he showed that evening. During the last year he had been endeavouring to obtain a series of dogs' skulls with the idea of acquiring a really good collection, as he thought they might from them gain some knowledge on questions relating to the etiology of irregularities of teeth. There were now something like sixty specimens, grouped under proper headings. The specimens he showed upon the table and by means of the lantern were the following:—

(1) A very interesting specimen of a rodent showing a curious wearing of the incisor teeth.

(2) A Japanese pug with a double cleft palate.

(3) A Rhesus monkey from India, showing destruction of the tooth tissue on the occluding surface of the mandibular right third molar. The alveolar process showed signs of periodontal disease, the destruction of bone being marked in the region of the mandibular molars. The mandibular teeth were practically free from salivary calculus, but slight deposits were present in the buccal aspects of several of the maxillary teeth.

(4) The skull of a bulldog showing in the mandible an absence of the first premolars—comparatively common amongst bulldogs—as well as the third molars, but there were signs that the right third molar had been present. Owing to the narrowness of the mandible compared to the maxilla the second molars occluded in a defective manner. The upper carnassial teeth were transverse to the arch and almost functionless.

(5) The skull of a bulldog showing the fourth premolar absent in the maxilla and the first premolars in the mandible.

(6) Another specimen of a bulldog showing still greater crowding in the region of the maxillary premolars. The premolar was absent on one side of the mandible.

(7) A specimen showing the malposition of the permanent canine owing to the retention of the deciduous teeth, a fairly common irregularity in dogs. Some examples of dogs and carnivora that were supposed to have two canines were very often cases of a persistent deciduous canine.

(8) A specimen of a toy Pomeranian showing advanced periodontal disease. Five of the maxillary incisors had been lost. In the mandible only three incisors were in position, and posterior to them and embedded in the bone near the median line were two cone-shaped teeth. The left first mandibular premolar was absent and the second premolar was misplaced and unerupted.

(9) A specimen of the woolly phalanger in which the pulp cavity of the right mandibular incisor had been exposed through attrition, leading to suppurative pulpitis and periodontitis. A sinus was seen opening below the mental foramen.

(10) A Barrasingha deer which died shortly after shedding its antlers. There were signs of periodontal disease, the destruction of the bone being most marked in the region of the first molars.

(11) A specimen of Campbell's monkey showing caries in the region of the third premolar. The specimen was interesting as showing a very curious condition of the two condyles.

(12) A specimen of a cod showing a large growth on the premaxillary bone. The growth was fully 1 in. across and was covered with teeth.

(13) A Blenheim spaniel with an extra incisor in the maxilla and the first premolars and first incisor absent in the mandible.

(14) A Japanese spaniel with the first premolars absent in the maxilla and the first premolars and third molars absent in the mandible.

(15) A French poodle with the left maxillary first premolar absent.

(16) Black and tan King Charles spaniel with the maxillary and mandibular premolars crowded and third molars absent in mandible.

(17) A Japanese pug with cleft palate and double hare-lip.

(18) A bull pup with double cleft palate.

(19) A red fox terrier with left, second and fourth mandibular premolars absent.

(20) A Mexican tree porcupine showing irregular wear of the incisor teeth.

(21) Retriever with extra tooth in the maxilla between the right first and second premolars.

(22) Bloodhound showing considerable destruction of the bone from periodontal disease, most marked in the region of the canines and premolars. The labial surfaces of the incisors showed marked abrasion, probably from rubbing the teeth against the bars of the kennel.

Notes on a Case of Extensive Necrosis of the Mandible.

By W. W. James, F.R.C.S.

H. W., a boy, aged eleven years and two months, who is present to-night, has a marked deformity of the face due to the loss of a considerable part of the mandible on the right side and from the remaining part being pulled backwards and to the right by the action of the muscles. The chin has receded and is displaced to the right. The right cheek and region of the angle of the jaw present a marked swelling, due chiefly to the displacement of the soft tissues, and partly to the inflammatory exudation, which is not yet completely absorbed. The teeth are practically normal except for changes which have resulted from the altered condition of the mandible. The left upper premolars which erupted in their normal position in the arch at the time of the first operation are now displaced inwards and articulate with those of the lower jaw. The lower incisors, which appear elongated are considerably inside the maxillary incisors.

The following history was obtained from the boy's mother: In April, 1906, he had a swollen face with severe pain; his mouth was foul, and the gums were ulcerated and so swollen as completely to cover two or three of the teeth. He was ill for about a week, had a high temperature, and at times was delirious. A medical man who was called in ordered a mouth-wash of borax and hot water. On the fourth day an abscess burst in the region of the first molar, the discharge being profuse and very foul. As his progress was unsatisfactory, the boy was taken to a dentist, who sent him to Guy's Hospital on the ground that he needed an extraction under gas. At the hospital the first molar was removed and a carbolic mouth-wash ordered. The boy became much better, but the discharge continued to come from the socket, and the

f—19*i*

swelling of the face did not diminish; he also had considerable difficulty in opening his mouth, and was unable to take solid food. In that condition I saw him at Great Ormond Street. He had very marked swelling of his face, with necrosis of the mandible extending from beyond the socket of the first molar as far forward as the canine and apparently to the lower border of the mandible. As a discharge of pus was coming up by the sides of the first premolar and the canine, they were removed and found to be necrosed, the pulps being dead. The mouth was cleaned as thoroughly as possible, and a mouth-wash at first of permanganate of potash, and later of liquor. potassæ and carbolic acid, was ordered. The condition of the mouth rapidly improved. The second lower molar had to be removed and was found necrosed; the second premolar was also removed, but this was found to have a living pulp. The treatment continued for about six weeks with the occasional removal of small pieces of necrosed bone, but the separation of the mass took place very slowly, as did also the formation of new bone, although just at the lower margin of the jaw it was fairly complete.

The boy's health during the whole time was very good, and it was possible to keep his mouth comparatively clean. I went on for several weeks with the hope of getting the sequestrum to separate, but as it did not do so I took the boy into the hospital and removed it. It was perfectly free in front, but at the back it was attached, not very firmly, to the bone. When this piece of dead bone had been removed I found quite a soft bed of granulation tissue. About a fortnight after the operation the wound appeared to be quite healthy and presented only soft tissues, the bone behind being completely covered. The day before the operation I had removed a tiny scale of bone which was projecting from the gum below the central incisors and was quite separate from the necrosed mass. I asked Mr. Arbuthnot Lane to see the boy with the view of operating to correct the deformity, which was very marked indeed. Mr. Lane thought that there was a possibility of being able to benefit him, and we suggested taking the boy into the hospital in order to arrive at a decision, but the mother was shy of the operation. I next saw the boy after about twelve months, when he came at the end of last year with a piece of necrosed bone projecting at the back part of the mouth on the right side. There was no marked discharge, but it caused him a little inconvenience. He was taken into the hospital, and I removed the remaining portion of the jaw. I operated through the mouth, making an incision to the outer side of the condyle, and, dividing the capsule, was able to remove the remaining piece of bone, which was quite necrosed. I made the incision from behind forward so as to avoid the structures lying behind the neck of the jaw. After removing the condyle, I made a careful examination of the articulation, which appeared to be quite destroyed; I could feel no sign of any dead bone or cartilage. I decided not to introduce any gauze, as I thought the position most favourable for free drainage. Everything went on well, and the boy is as you see him now. On one day I was a little anxious when he had a temperature of 101° F., and I found that the retracted position of the jaw, by kinking the sinus, had caused pus to become pent up. Since then, by depressing the jaw and keeping the sinus freely syringed, there had been no further trouble.

One of the remarkable things about the case is that the patient appeared to be upset very little indeed, except at the time of each of the operations; otherwise neither his temperature nor pulse-rate was raised. It is rather curious that we do not get more cases of this kind. One point not quite clear is whether the condyle was actually dead at the time I did the first operation. I do not think it could have been, though it seems strange that the condyle should afterwards die. The separation of the first sequestrum, the lack of any discharge, and the covering up of the bone lead me to think that the necrosis was not present at the time I removed the first sequestrum.

I should like to know the opinion of members of this Section as to whether it is wiser to wait for new bone to form round the old sequestrum before removing it or to endeavour to give the patient a clean mouth by removing as much of the necrosed bone as possible. If the former method is the better, I ought perhaps to have operated later; if the latter, I ought certainly to have operated earlier.

DISCUSSION.

Mr. F. COLEMAN said he had recently seen a somewhat similar case to that referred to by Mr. James, but occurring in a man, aged about 30, the sequestrum extending from the left wisdom tooth to the right canine, involving the entire sockets of the molar tooth and the inner alveolus of the remaining teeth. When he saw the patient the sequestrum was almost loose. He waited a few weeks until it had separated and was practically embedded in granulation tissue with no bony attachment and then removed it under nitrous oxide; it came away quite easily. There was no fresh bone opened up to infection. A fragment of bone projecting into the floor of the mouth was removed a few days previously, and the patient still had some necrosed bone, which was being carefully watched. The piece of bone came away cleanly and left a nice granulating surface. He should always feel inclined to wait until the sequestrum was loose if the mouth could be kept reasonably clean and there were no indications to the contrary.

Mr. J. H. BADCOCK asked whether any attempt had been made, by means of a splint, to hold the mandible in position while new bone was being formed.

Mr. W. B. PATERSON corroborated what Mr. Coleman had said with regard to his case. His own impression with regard to that case was that it was caused by a dirty instrument in the extraction of a lower molar. There was a very sloughy, dirty-looking condition of the gum when the man first came before him a few days after the operation, and there were certain points about the operation and the operator which he would not further allude to, but which helped his diagnosis. With regard to the question of the time of operation on a sequestrum, raised by Mr. James, more than once it had occurred to him that some good might come from gouging, and in Mr. Coleman's case he urged him to an operation before the sequestrum was loose. Under the anæsthetic some force was required in the removal of the sequestrum. He admitted it was against the principles laid down in the books on surgery, but it had the advantage of removing a great deal of dead and infective matter and rendering the mouth more hygienic, and if there was a small amount of sequestrum afterwards it might be more easily dealt with. On the other hand, in dealing with cancellous bone there might be the danger of starting an inflammatory condition. He should like a little further history of the case Mr. James had presented, both with regard to operation and instruments in the extraction of the molar. He asked whether arsenic had been used in any previous treatment. He had seen bad cases of necrosis following arsenic. The case was interesting in showing such a tremendous loss of bone, and differed from Mr. Coleman's case in being only on one side of the jaw.

Mr. W. W. JAMES, in reply, said he had considered very carefully the possibility of maintaining the jaw in its proper position by a splint: in fact, he had considered many methods, and had hoped that something would have been said that evening concerning the correction of the deformity. With regard to the stage at which the necrosed bone should have been removed, as the amount exposed was extensive and the boy's mouth foul, he thought it wiser to operate than to wait. He thought it should be the rule to remove dead bone, although, if of considerable amount, whether it actually affected the bone that remained it was difficult to say. With regard to the history of the molar the only thing he knew was that the boy heard the student who removed the tooth at Guy's Hospital remark that it was a "dead tooth." He did not think that it had ever been filled or attended to by a dentist; and he did not think arsenic had been used. He once saw a case similar to that described by Mr. Coleman. It was that of a navvy who came to the Dental Hospital, Leicester Square, to have what he called a "big tooth" removed. It was found that he had necrosed bone extending from the region of the third molar to the canine tooth, and the bone was removed. He said he had had some trouble with "the tooth," as he called it, after the extraction of another tooth. The disease had apparently followed extraction. A remarkable point about this case was that the man had never been away from work for one day.

The Treatment of Children from the Dental Aspect.

By J. F. Colyer.

In glancing through the *Transactions of the Odontological Society* we cannot fail to notice the scant amount of attention that has been given to the important question of dental disease in children. Two papers I can recall, namely, one by Mr. Edmund Owen in vol. xxvi., p. 194, and one, more recently, by Mr. Denison Pedley.

This absence of papers on the teeth of children is all the more inexplicable when we consider the prevalence of dental disease in childhood, and also its important bearing on the future health of the individual. Under these conditions I have no hesitation in drawing your attention to-night to the subject. I do not pretend that in the remarks I shall make you will find anything new, but I wish briefly to discuss and review certain aspects of the question, in the hope that they may stimulate enquiry into some of the varied problems that are awaiting solution.

In reviewing this problem of dental disease in children we must try and consider it in its broadest aspects, and look at it, not only from the point of view of those able to pay for skilled treatment, but also from the view of those whom our defective social system compels to seek aid at the hands of charity. Regarded from this broad point of view, the first thing that strikes me is the appalling amount of disease and the impossibility of dealing with it by curative measures. Take London alone—it is computed that there are something like one million school children, and of these probably 90 per cent. require treatment—and you have some idea of the magnitude of the task. To discuss the method of dealing with the teeth of the children in our State schools would occupy more time than we have to-night. The only remark I would make is, that looking at the problem as it presents itself to me in the course of my duties as a dental surgeon to a public institution, I am convinced the only method of tackling the question is by concentrating attention on the prevention of disease, not so much by instruction to the children as by instruction to the parents. In the majority of papers one reads on the subject one is very much struck by the importance placed on tooth brush drill and care of the teeth; but it is not much good teaching children how to clean their teeth if you do not instruct the parents to see that the thing is done.

To prevent disease we must first ascertain the cause, and this brings me to my first point. Do we thoroughly understand the cause or causes that are at work producing the terrible amount of dental disease we see? I think the answer must be in the negative.

It is now over twenty years since Dr. Miller's epoch-making work was published, in which he demonstrated the pathology of caries; but the pathology of a disease is not the etiology, and we still, I venture to think, have by no means a clear conception of the cause or causes at work. Fortunately, during recent years, more attention has been given to the subject, and out of the dim haze of facts light is appearing.

Let us for a few moments consider some of the debatable points in connection with this question of etiology. Mr. Hern, in discussing a paper by Mr. Dolamore[1] on the condition of the teeth of the populace, tells us that "the work of Miller was an epoch-making one when he showed that the main cause of dental decay was the resting and fermentation of carbohydrate food on the teeth." I quote this remark because a large number of practitioners are content to consider this an ample and satisfactory explanation of the cause, and on this have based their preventive treatment, which consists in the removal of food particles from the teeth by artificial aids, such as the tooth brush and silk. These artificial aids have naturally lessened the amount of decay; but throw them aside, and you will find yourself just as powerless to prevent decay as you were fifty years ago. Particles of carbohydrate food rested on the teeth of our ancestors, rested on the teeth of prehistoric man, but decay of the teeth did not occur. Semicivilised peoples of to-day eat carbohydrate food, but decay amongst them is rare.

It is quite clear, then, we must not rest content with such a superficial statement as explaining the etiology of decay, but we must, I think, look more fully into the character of the food we eat to-day. Most excellent work has been done in this direction during the last few years by Dr. Sim Wallace, and his view that the ridding of the foodstuffs of their fibrous parts by methods of preparation renders them more liable to lodge about the teeth is probably a most important factor. A point in relation to the foodstuffs to which attention has been drawn by Mr. S. Colyer is the question of the increased fermentability of the carbohydrates now used, and also the introduction of monosaccharides. I think that the increased importance given to sugar as an article of diet is, in a great measure, accountable for much of the caries of children. Dr. Miller and others did not attribute much importance to the part played by sugar in causing caries, holding that it was rapidly dissolved in the saliva and so passed away from the mouth.. These observers seem to have overlooked the fact that, although the sugar may be dissolved by the saliva it may be retained in the fissures on the occluding surfaces, and also, what is more important, by capillary attraction around the points of contact of the teeth. Specimens illustrating this latter point are occasionally to be seen in some teeth removed for the purpose of correcting crowding of the teeth. At the point of contact of the teeth there is a little island of enamel, quite free from caries, surrounded by a zone of decalcified tissue. If we recognise this capillary attraction, then the harmful action of sugar in solution is easily understood. In connection with this question of sugar we must realise that an enormous amount of the sugar eaten is in the form of glucose, and *not* cane sugar. The former is a monosaccharide, and therefore easily fermented; the latter a polysaccharide, and therefore requires to be "inverted" before being capable of undergoing fermentation. That is, to my mind, a very important point in the question of the etiology of caries.

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[1] *Brit. Dent. Journ.*, 1905, xxvi., p. 1.

On these theoretical grounds, and also from clinical experience, I am convinced that not half sufficient attention is paid to the use of sweets. We may fill teeth, and our patient may carry out the rules laid down for cleaning, but if the use of sweets in any quantity is continued our labour is in vain. Much is written about alcohol as a factor in physical deterioration: I venture to think that the harm done to the race by the constant eating of sweetstuffs runs alcohol very close. I know my view about the use of sweets by children is not a view generally held by practitioners, but such glaring examples are met with in practice of boys who eat sweets at school and come back with caries time after time that the matter should be considered. If the boys are stopped from eating sweets they come back from school with their mouths pretty free from caries. I think the teaching that sugar, because it is dissolved, is rapidly swept away by the saliva is bad teaching, and that sugar has been underrated as a cause of caries.

The whole question of the prevalence of decay is really a question of the last fifty years. Caries has become much more prevalent within that time. If you look very carefully through the question of foodstuffs you will find that during that period the foodstuffs responsible for causing caries, the carbohydrates, have undergone an extraordinary amount of variation. An example of the altered character of our foodstuffs is to be found in the flour of the present day. There is evidence to show that the roller milled flour is more agglutinative and more acid-producing than stone milled flour. The question requires very careful and thorough investigation, and is of great importance from a national point of view. I instance flour because it is a good example of a foodstuff having undergone radical changes in its character in the space of a generation. Still further, it must be remembered that flour forms a most important item of the food of the poor. This point is brought out very clearly in the volume issued by the Board of Trade on the cost of living of the working classes. It is found that for incomes below 25s. per week two-thirds of the amount is spent on food, while of incomes of 40s. the amount is about 57 per cent. In incomes below 25s. about 21 per cent. is spent on flour and bread; for incomes between 30s. and 40s. the amount is about 15 per cent., the actual quantity of bread and flour purchased varying from 28½ lb. to 37¾ lb., with an average of about 32 lb. In families earning less than 25s. per week altogether about 58 lb. of food is consumed (leaving out milk, which is about 5·5 pints), and about half of this is flour or bread. Therefore with the working classes flour is a very important item of diet, and its method of preparation does seem to me to want thorough investigation as far as this question of caries is concerned.

Before passing on to consider the application of the food question to the prevention of caries, I feel I must run a tilt at a teaching that has gained ground during recent years, namely, that the structure of the tooth plays but little part in the question of caries. How such a doctrine could have gained ground on the inconclusive investigation carried out to me is inconceivable. Clinical experience shows conclusively that the teeth do vary in their resistance, and this resistance is, in all probability, due to the qualitative, and not the quantitative character of the salts entering into their formation. The physical character of the salts probably plays an important part, and the varying hardness possibly depends on this fact. In geology a pretty fair example bearing out this point occurs in the forms of calcium carbonate met with in Nature. The two forms, calcite and aragonite, vary in their hardness and resistance, the main difference between the two forms being the method of crystallisation: calcite, the harder, belongs to the hexagonal system; aragonite, the less resistant, to the orthorhombic form. Further, with regard to this point I think it is a fatal mistake to jump to conclusions. We jumped to this conclusion about the structure of teeth on the strength of certain investigations made by Dr. Black, and those investigations were made mainly on dentine. They were simply quantitative and not qualitative investigations, and on that the dental profession—and I am sorry to say some of our teachers—have taught that the question of the structure of the teeth bears no part in this question of decay. It seems to me that the teeth must bear a very important part in the question of resistance to attack. Although the teeth may not vary in the actual quantity of the lime salts in them, they may do in the quality, and this may depend on the way in which they are laid down in the organic structure of the tooth. In this connection a point that requires investigation is the influence of food and methods of feeding on the formation of the teeth. Dr. Kingston Barton, who has kept accurate records, is of the opinion that breast fed children have better teeth than those that are hand fed. Other observers maintain that, providing the hand feeding is properly carried out, no difference can be observed. There is, however, ample clinical evidence to show that hand fed children, brought up on condensed milks, patent food in which starch is an important factor, do often possess defective teeth. The early feeding must affect the structure of the tooth, but where the fault lies has yet to be determined. Then there is another problem awaiting solution in the etiology of caries, namely, the amount of lime contained in the food. Evidence collected by Dr. Rose goes to show that the amount of lime bears an important relation to the incidence of caries.

I have referred to these points in connection with etiology to show how difficult it is with such imperfect knowledge to formulate a line of preventive treatment which will be absolutely reliable. Our knowledge of the etiology would seem to indicate that preventive treatment should follow on somewhat the following lines:—

(*a*) The insistence of breast feeding.

(*b*) The use in early years of foodstuffs which require efficient mastication.

(*c*) The insistence of mastication by the child.

(*d*) The use of carbohydrates which are not easily fermentable. Sweets in the form of sweetmeats should be forbidden.

As secondary in importance to the above, the proper use of the tooth brush, especially after the last meal of the day. If parents' attention were drawn to these points, and the directions faithfully carried out, I believe that 75 and 80 per cent. of the dental disease we see would disappear.

With regard to the curative treatment, we must always keep clearly in mind the necessity of rendering the mouth functional. Unless this is done, no amount of tooth brushing work will keep it clean. The most important point to keep in view is that children's mouths must be rendered functional, and if that is done they will probably be kept clean.

First, then, I would insist upon the importance of proper nasal breathing. Oral breathing tends to persistent gingivitis of the gums in the front of the mouth, with the sequelæ, caries, suppuration, &c. In the past the part played by oral breathing in the production of a dirty mouth has been under-estimated: but of the important part nasal breathing plays in rendering the mouth functional there can be but little doubt. I might mention in this place a sign of mouth breathing that is often overlooked—namely, a slight gingivitis of the gums covering the incisors, the gums at the back of the mouth being healthy. I regard this sign as almost diagnostic of mouth breathing.

With regard to conservative treatment, much may be done by filling, providing the pulp of the tooth is not exposed. In cases where the pulp cavity is exposed I am inclined to think that extraction is nearly always the better line of treatment. Indeed, the only instances where one is inclined to adopt conservative treatment are in the cases of second deciduous molars in children under the age of 6—in other words, in cases where the first permanent molars have not erupted.

I fully appreciate the fact that the large majority of practitioners do not agree with such radical treatment, but the impossibility of thoroughly treating pulp chambers in children and the all too frequent subsequent suppuration inclines me to the opinion that by extraction we are more likely to ensure the mouth being rendered functional.

The cases, however, which demand most serious consideration are those so commonly seen in hospital practice, where child after child is brought for treatment with the majority of the deciduous molars hopeless wrecks. If the first permanent molars are in position, the best treatment seems to be the removal of all the deciduous molars. This has the advantage of removing all sources of sepsis and of isolating the first molar—a point, to my mind, of the greatest importance when one considers the value of this tooth in mastication. I do not hesitate to remove sound molars if their antagonists have been removed. Perhaps I can make my view more clear by giving one example. Suppose the right maxillary deciduous molars and the left mandibular molars are unsavable and their removal is called for, then the remaining teeth—namely, the right mandibular and the left maxillary molars—are rendered functionless, and can serve but little good purpose; indeed, they will harbour food, and so prevent the mouth being kept naturally clean. The removal, then, of all deciduous teeth not functional is called for if we hope to render the mouths of children clean.

It will be urged that such treatment robs the child of masticating power, but such teeth are useless as far as the function of mastication goes; indeed, they render the first permanent molars functionless, because a child with tender teeth "bolts" its food and cannot chew.

It will be urged that the removal of the deciduous molars allows the first permanent molars to come forward, and so cause in the future crowding of the anterior teeth. With regard to this, I would point out that such travelling forward of the permanent teeth occurs mainly in mouths where the growth of jaws is interfered with by want of function, either due to insufficient mastication or lack of nasal breathing. From observation—but I express myself guardedly in this respect—I am inclined to think that if, by removing the deciduous molars, you can render the first molars functional, the growth of the jaw will be stimulated and room made for the development of the second and third molars, with no forward pressure from these teeth, and, with the first molars occluding correctly, there will be little, if any, forward movement.

But, granting that the treatment of extraction suggested does cause a moving forward and subsequent crowding, the removal of four teeth will easily alleviate the condition. One has to weigh in the balance the loss of four teeth against the constant presence of oral sepsis and all its sequelæ. In my opinion the former far outweighs the latter.

Still further, one must always remember that even if the deciduous molars were retained with the object of preventing the moving forward of the molars, it is more than possible that the removal of both to relieve crowding would be required, as in such an individual some interference with the development of the jaws would probably have taken place. In children, where the first molars have not erupted, the removal of the sound antagonistic teeth should be postponed until the permanent teeth have erupted, but no hesitation should be made about the removal of deciduous teeth that cannot be rendered aseptic.

In cases where the deciduous teeth are decaying on all surfaces, such as occurs from the sucking of sugar-bags or the constant presence of an easily fermentable carbohydrate on the surfaces of the teeth, a considerable improvement can be obtained by careful regulation of the diet and cleansing of the teeth, combined with local treatment of the teeth. For the latter purpose no drug acts better than nitrate of silver, and this should be applied to all the carious surfaces of the teeth at least once a week until the denture shows signs of hardening. The arrest of the condition can also be applied by the regular use by the parents of spirits of wine and an alkali.

I admit that the above idea of the treatment of children from the point of view of extraction is probably novel, but I should like to take those who have not tried it to the Royal Dental Hospital. It is really remarkable to see children, with possibly only three or four incisors, perfectly healthy simply because there is no oral sepsis. If we want to have a child healthy its mouth must be clean, and the mouth cannot be clean if it is a mouth breather or if it has one little root in its mouth that will prevent it eating. It is very dreadful the way in which practitioners of dentistry will not attend to children. If we are going to do anything for the race we must look after the children. It is the children who will make the race in the future, and we must attend to them. Still further, we must realise that we do not know the cause of decay, and the best thing is to set about the matter and find it out.

DISCUSSION.

The PRESIDENT (Mr. Howard Mummery) did not think that Miller minimised the effect of sugar upon the teeth: he simply drew attention to the fact that starch was just as injurious to the teeth as sugar, showing that the lactic acid fermentation was set up just as soon when sugar was taken into the mouth in the form of starch as when taken as sugar, but that when cane sugar was taken into the mouth it was more quickly dissolved away. Miller drew attention to the caries amongst confectioners and bakers, where sometimes the central incisors were destroyed by sugar dust. On the question of lime salts Miller did not think that Dr. Black's conclusions were at all conclusive, because it depended upon the mode of combination of the lime salts, and they might be combined loosely or compactly. As far as Dr. Rose's investigations were concerned, Dr. Rose had not yet come to any definite conclusions, as he had had a great many contradictory results.

On the motion of Mr. W. HERN, the discussion on the paper was postponed until the next meeting on February 24.

Odontological Section.

February 24, 1908.

Mr. J. Howard Mummery, President of the Section, in the Chair.

The Dental Uses of Paraform.

By J. E. Spiller, L.D.S.

Paraform, a white amorphous powder with a pungent odour, is a polymer of formaldehyde. It has two principal uses in dental surgery; as an obtundent of sensitive dentine and as a dressing for root canals. As an obtundent it may be used in suitable proportions with almost any temporary filling material. Personally, I find one of the most convenient methods is to mix oxysulphate of zinc (which is practically the same as the powder of artificial dentine) and paraform in the proportion of 100 parts of the former to 5 of the latter; this is mixed with an aqueous solution of gum arabic to the consistency of a stiff paste in the usual manner. The whole cavity may be filled with the mixture or, if slight separation is required, the cavity is lined with it and then tightly plugged with base-plate gutta-percha.

In similar manner paraform may be mixed with gutta-percha, mastic, zinc phosphate or any of the temporary filling materials. The dressing should be left from one to seven days, but may be left without danger for a longer period if unavoidable. At the end of this time the cavity may be cut with practically no pain in nearly every case. Pain occurs from the dressing in a small percentage of cases, and is usually not localised in the tooth under treatment and may be referred to almost anywhere in the maxillæ of the same side. It is seldom severe and does not last long. The insensibility does not extend to the pulp, the extent to which it penetrates usually being about equal to the thickness of a thick postcard. (This of course varies according to the strength of the paraform in the dressing and the length of time it is left in the cavity.)

This, I think, proves that the action of the drug is confined to the dentinal fibrils.

Paraform being an irritant should not be placed in an aching tooth or too near the pulp; if the cavity under treatment is a deep one the mixture containing the paraform must be confined to the more superficial parts and the remainder of the cavity filled with other material. By this means one is able to shape a cavity with an inverted cone bur where previously the dentine was acutely sensitive, even when touched with cotton-wool or a steel probe.

As a dressing for septic root canals it may be mixed with any of the essential oils into a thin paste and applied on cotton wool. It is especially valuable in canines or posterior teeth where a sinus exists, as in this case the canal is rendered sterile and the sinus invariably disappears within a few days. Mixed with zinc oxide, thymol and other substances it is undoubtedly the essential constituent of most of the proprietary substances sold as "abscess cures."

Paraform may be incorporated with the permanent root canal filling; if the tooth has been devitalised, 1 per cent. of paraform in the filling is sufficient; if the root canals have been septic 5 per cent. or more should, I think, be used. Paraform may also be used in very small quantity to mix with the cement used for setting crowns, to keep it sterile (in larger quantity it interferes with the proper hardening of cement) and for devitalising shreds of pulp where arsenic has failed.

Personally I have used paraform in the manner briefly described almost daily for four years, and frequently many times a day. During this time I have, to my knowledge, devitalised one pulp only, in which case I afterwards found that there was a minute exposure of the pulp. In no case can I record a failure except where the dressing has failed to hold. In a case where there is a probability of this occurring the dressing and the whole side of the tooth should, if practicable, be covered with Harvard cement.

DISCUSSION.

Mr. H. BALDWIN corroborated Mr. Spiller's statements and expressed his pleasure at the subject having been brought forward. Paraform was a remarkably fine antiseptic and was the most useful obtundent ever introduced. Until its introduction the *bête noire* of dental surgery was the sensitiveness of the dentine, but now by its use the sensitiveness could be reduced practically to nothing. The only disadvantage was its irritating properties, although they were nothing like as bad as ordinary formalin. The proportion to be used required carefully estimating, and, as Mr. Spiller had pointed out, the nearer the cavity was to the pulp the smaller the proportion to be used. The most important use of paraform was as an obtundent, and to Mr. Spiller was due the credit of introducing it for that purpose. It was an extraordinary drug, in that the formaldehyde which paraform evolved appeared to be capable of penetrating almost anything. The drug could be used intimately incorporated with gutta-percha or mixed with Harvard cement, and could be also mixed with gum sandrac varnish as an ordinary dressing. This property was most useful because Harvard cement could be made to stick in superficial cavities in the necks of teeth with practically no excavation, and when mixed with, say, 5 per cent. of paraform made a temporary stopping which would quite desensitise these troublesome cavities in a very few days.

Mr. WILLIAM HERN said he was able to confirm much that had fallen from the two previous speakers in respect to paraform: the drug was introduced to his notice by Mr. Baldwin. He had used it for some time and had found it a valuable obtundent for sensitive dentine, but it should not be experimented with except in a very dilute solution. He began using it a little too strong and the first two patients he treated for sensitive dentine with what he thought was a trace of paraform returned to him with "toothache."

Mr. GABELL thought that any caustic drug might produce an obtundent effect, and so also would any efficient protection of the cavity from sepsis and irritants by an inert dressing, and he wished to know how much of the obtundent effect of the paraform was due to the drug and how much to the dressing.

Mr. A. E. BAKER had also used paraform for some time and found it one of the best obtundents that had come under his notice. He asked whether Mr. Spiller could explain its action upon the dentinal fibrils. Very often cavities could be cut out with no more sensation than cutting out a piece of wood. He had had one or two mishaps. In a lower molar with caries on the bucco-cervical region great pain was experienced. He removed the paraform dressing, cured the pain by other means and was eventually able to cut out a cavity quite satisfactorily, and some six months after, on testing the teeth, he found the pulp alive, and it was alive to-day.

Mr. E. B. DOWSETT did not think Mr. Spiller laid sufficient stress on the time the paraform should be left in. He had used it for about two years or longer, and his experience had been that with a paste composed of a mixture of paraform one part and zinc oxide ten parts the longer it was left in the better. It should be only just a smear. He had left it in one cavity as long as six weeks, and it was the most successful cavity he had operated upon. That may have been due to the sealing of the cavity for so long a time, but, on the other hand, cavities long sealed with gutta-percha alone had been found more sensitive when opened than they were before sealing.

Mr. J. E. SPILLER, in reply, said he was afraid he could not claim the introduction of paraform as an obtundent, as it was originally suggested to him by

Dr. Mahé, of Paris. He was filling some of that gentleman's teeth and was asked by him to use a little paraform powder mixed with zinc oxide and a liquid composed of five parts of menthol crystals and four parts of carbolic crystals. They were made into a thin paste with which the cavity was smeared and then filled with gutta-percha. If mixed with liquid there was more certainty of an even mixture than with powder. Bad results from paraform were often caused by a spot of paraform not intimately incorporated with the zinc oxide. With regard to the quantity, he agreed that 5 per cent. was quite sufficient. In reply to Mr. Gabell, he thought nearly everything was due to the drug and very little to the sealing. When carbolised resin, covered with gutta-percha or other dressings, had failed to decrease the sensitiveness and he had put in paraform he had made the cavity almost completely insensitive, a fact which he thought proved that it was due to the action of the drug and not to the sealing of the cavity. It was quite true that a pulp did not always eventually die under an obtundent; it did not die under nitrate of silver, for instance. Several of his own teeth had been dressed and the pulps were still alive. If paraform was put into a cervical cavity, for example, and left in, it would be found that a distal or crown cavity was as sensitive as before, showing that the action was purely local. It was a difficult matter to explain the action of the drug, but he believed it was probably due to the formalin hardening the dentinal fibrils. It would harden to some extent decalcified dentine. He thought the hardening action was a very useful one in root canal dressings or when used as root canal fillings. As regards the time it was left in, that should be, he believed, in inverse ratio to the strength of the mixture; a very weak mixture might be left in for a long time, but he should not care to leave in a 10 per cent. mixture as long as six weeks.

On the Termination of the Nerves in the Teeth of Mammalia.[1]

By W. J. Law, L.D.S.

No apology is necessary for the reopening of the discussion, time worn though it is, upon the mode of the final termination of the nerve fibres found in the pulps of the teeth of mammalia, for in spite of all the work that has been done upon this subject by very numerous histologists no solution of the problem has as yet commended itself to the majority. At the present time we find that broadly speaking they may be divided into two classes: first, those who think that the fibres after leaving their medullary sheaths actually enter the dentinal tubules and, running in them, terminate in the granular layer of Tomes or at the amelo-dentinal junction; secondly, those who deny that they enter the tubules but believe that they terminate in or around the odontoblast cells.

Before, however, we enter upon such an investigation there are certain a priori considerations which have to be clearly understood for an intelligent grasp of the problem that lies in front of us. The most important of these is the sensibility of dentine. We as dentists are, alas, only too familiar with this, and there are few, if any, of us who doubt that this is a true sensibility and not a mere effect of pressure transmitted mechanically through to a sentient pulp. If any doubt exists it should be cleared by the fact that the sensibility of dentine is greatest at its periphery, and that it can be stimulated by such chemical reagents as dilute acids, *e.g.*, phosphoric.

We must all of us have noticed that while the introduction of oxy-phosphate cement is accompanied by no pain in the majority of cases, in those cavities which are very sensitive to excavate, many of which are very shallow and so less liable to transmit thermal changes, such introduction is accompanied by pain of an acute though temporary character. If, then, we turn to physiology for an explanation of this fact we find that so far as is known all sensation in all parts of the body is produced by the direct stimulation of afferent nerve fibres, and thus by inference we should expect to find nerve fibres in the dentine of human teeth at any rate. This is a conclusion that I do not think has been fairly faced by those who deny their presence in dentine; thus Carl Huber, of Michigan, gets out of the difficulty by denying the sensibility of dentine. He, however, is not a dentist, and I can only conclude that he must himself have either a very good or a very bad set of teeth which have not up to now entailed many filling operations. Hopewell-Smith, while admitting the sensibility of dentine, thinks that it is due to the processes of the odontoblast cells acting as "sensation transmitters," or nerve end organs. Although there can be no doubt that processes from the odontoblasts do enter the dentinal tubules, the difficulties in the way of accepting this view seem to me to be even greater than in Huber's case, for it is to be noted that in the few cases of such nerve end organs as we know, the cells that play such a part are derived from the same developmental layer as the nervous system itself—the epiblast—while all investigation up to now has tended to show that the odontoblast layer is derived from the mesoblast. There is, however, an important exception which may seem to militate against the general truth of this statement: the case of skeletal muscle. Skeletal muscle undoubtedly conveys along its length, when excited, a process which is analogous to the propagated condition of excitation characteristic of nerve and spoken of as the nervous impulse.

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[1] A grant towards the expenses of this research was made by the Odontological Section of the Royal Society of Medicine.

Muscle, therefore, a mesoblastic tissue, is capable within its own boundaries of behaving like a nerve. This exception, however, only accentuates the position taken above, since the muscle is strangely unable to propagate any excitation beyond itself. Thus the excitement generated in a muscle by direct stimulation of unnerved portions, though travelling throughout the length of the muscle, does not pass into the nerve fibres supplying the muscles nor into any adjacent nerve fibres whatever. Langley has worked this point to some nicety, inasmuch as he has shown that nicotine applied to muscle in the neighbourhood of the nerve ending, and in fact by exciting the muscle itself in this neighbourhood, does not in any way affect the nerve fibres. Muscle, therefore, is incapable of doing what the odontoblast by this hypothesis is assumed to do, since it cannot affect neighbouring nerve fibres. The motor muscle, end plate or nerve ending is, by reason of some unknown quality, a perfect valve allowing a transmission of excitation across it in one direction only, from epiblast to mesoblast. The opposite mode of transmission, from mesoblast to epiblast, is unknown. Nor can this be explained by attracting attention to any intrinsic difference in the nerve fibres in these two cases, muscle and odontoblast. In the case of the muscle we have discussed its relation to efferent nerve fibres; in that of the odontoblast the hypothesis suggests a connection with afferent nerve fibres habitually accustomed to convey impulses towards the central nervous system. Both sets of nerve fibres, however, have the common quality that when once excited at a point they can convey this excitation in both directions; if the muscle did excite its efferent fibre that excitement would be conveyed. The point is that the muscle does not succeed in accomplishing this initial excitation, and there is similarly no ground for supposing that the odontoblast would succeed in doing what the muscle fibre cannot.

Such a theory of the function of the odontoblast cell either postulates a condition of things differing essentially from that found in any other portion of the body, and so only to be received with the utmost caution and upon the most incontestible evidence, or must be accompanied by proof that the odontoblast cell is an epiblastic structure. Such proof has never been attempted. The argument and long list of reasons for assuming such nervous function given in the paper[1] do not seem to me to be of the necessary weight. Thus "the axiom that dentinal fibres are considered to be sensation conductors is well known to all" is very much like begging the question, for it assumes as an axiom the very fact he is labouring to prove, and so the rest of the argument: "inasmuch, then, as they are regarded functionally in the light of nerves, and as they represent the peripheral poles of the odontoblasts, and are in fact part and parcel of those cells, it follows that the latter must be concerned in the act of conveying extrinsic stimuli to the nerves of the pulp," again assumes that the odontoblast processes are the *only* contents of the dentinal tubules—which is the point in dispute. Again and again does this author, in his reasons for doubting the dentine-forming functions of the odontoblasts, assume that they are concerned in sensation and then conclude that as they do not form dentine they must be sensation transmitters.

In the second place, this theory is inadequate to account for the varying sensibility of dentine, which ranges from almost complete absence to the sharpest of sensations. Is it seriously suggested that it depends upon the number of dentinal tubules?

Lastly, it must never be forgotten that negative evidence is at the best inconclusive, and that even one small piece of positive testimony is worth many larger pieces upon the other side, and that observers such as Oscar Römer have stated that they have seen the non-medullated fibres actually passing into the dentinal tubules; so that although the famous example of the true nature of the Nasmyth's membrane must make us very cautious about accepting a priori or analogous reasoning, it seemed necessary first of all to work upon the assumption that the fibres do actually enter the tubules as the most likely theory.

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[1] *Trans. Odont. Soc.*, 1893, xxvi., p. 9.

The literature upon the subject is mostly connected with the names of Morgenstern and Oscar Römer. The work of Morgenstern may be very briefly dismissed. Working by Golgi's method he concluded that all black staining was due to the presence of nerve fibres, and found several present in each tubule. But we know that silver nitrate alone will stain Neumann's sheaths, and I think that the thin black lines seen by him are an optical illusion caused by diffraction images of the cut walls of the tubules. Fig. 1 illustrates this. It is from a ground section of a human tooth which has been placed in silver nitrate in the dark, and then developed in a pyro developer in sunlight. Not only did Morgenstern fail to show that these black markings

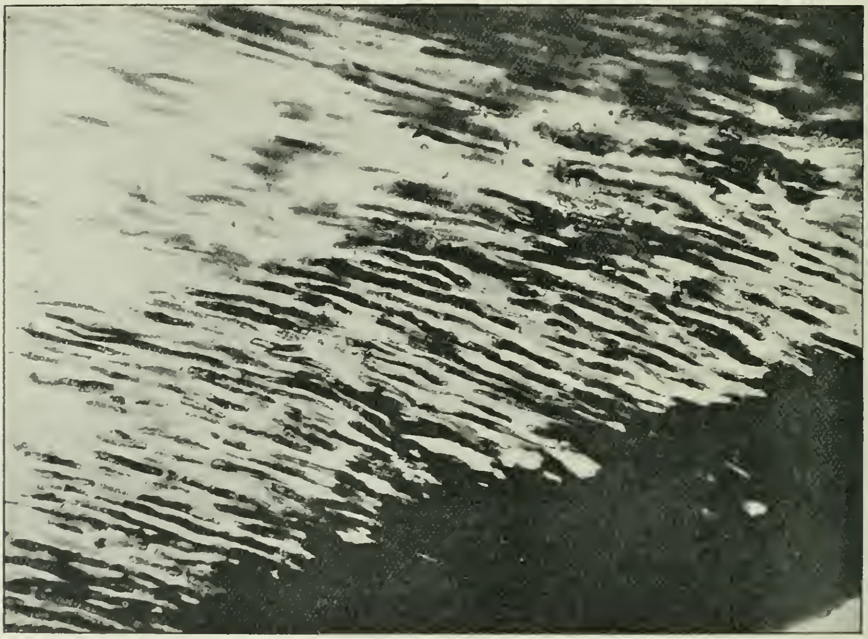

Fig. 1.

Human Incisor. Silver nitrate. Showing Morgenstern's neurofibrils (?) × 1760.

proceeded from the nerve fibres in the pulp, but he threw what I believe to be undeserved suspicion upon Golgi's method used in connection with the investigation of teeth. Oscar Römer's work, on the other hand, cannot be so lightly dismissed. Using the intravitam method of staining with methylene blue he figures the fibrils as entering the odontoblast cells and proceeding in them as far as the granular layer of Tomes. There is nothing unlikely in this, as it is strictly analogous to the method by which the axis cylinder obtains its medullary sheath.

"Each nerve fibre develops as an independent outgrowth from a nerve-cell and grows distalwards, finally becoming united to other tissues (*e.g.*, muscle fibres) in the periphery of the body. At first the axon is naked throughout its entire length, but later in development it becomes sheathed" is the most favoured view of modern neurologists, as against the idea that the nerve fibres are composed of the specialisation and linking up of cells already present in the peripheral parts of the body.[1]

The principal reason why Römer's work is not more universally accepted seems to lie in the fact that he made use simply of drawings to illustrate his argument. Not having had the opportunity of examining his slides, I am unable to express any views upon his work save that, as I have mentioned before, a piece of positive evidence of this kind seems to me to carry much more weight than many more apparently satisfying researches with negative results. One rather suspicious result of Römer's is that he did not find nerve fibres at the neck of the tooth, which we know from experience is usually the most sensitive spot.

In the attempt, then, to demonstrate the presence of nerve fibres in dentine all the most modern methods of staining nerve were reviewed to find the most suitable for use in the special processes concerned in the obtaining of sections of teeth with both the hard and soft tissues in situ. During the last few years much attention has been paid to the demonstration of nerve fibres in all parts of the body, and many new and improved processes have been invented for the purpose. At the present time the principal methods in use are the methylene blue and those associated with the names of Golgi, Ramon y Cajal and Bethe. Owing to the great difficulty of obtaining thin sections of teeth with the hard and soft tissues in situ without decalcification I have not been able to get any successfully stained specimens by the methylene blue process, the acid used destroying the stain. Ramon y Cajal's method of staining with silver nitrate and developing with a photographic developer has also up to now been unsuccessful in my hands; but the method of Bethe seemed almost ideal. His process is long and complicated, but as it may not be generally known I append a description of it, as varied for use with teeth:—

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[1] See Halliburton, "The Repair of a Nerve," *Science Progress*, January, 1908.

Small pieces of perfectly fresh tissue are fixed by placing upon blotting paper and covering with a 10 per cent. solution of commercial nitric acid. This serves to decalcify as well as to fix them and also lessens the susceptibility of Nissl's granules to take the stain. They are left in the acid until decalcified (forty-eight hours) and the acid is frequently changed so as to keep it of as uniform a strength as possible. They are then placed in 8 c.c. of alcohol, 90 per cent., 3 c.c. of water, and 1 c.c. of ammonia for twenty-four hours. If they turn brown, discard. This is due to impure nitric acid or too long immersion. Again place in alcohol for six to twelve hours, then in 1 c.c. of HOl, 3 c.c. of water, and 8 c.c. to 12 c.c. of alcohol for twenty-four hours. Then alcohol again for ten to twenty-four hours, distilled water for two to six hours (not longer), ammonium molybdate, 4 per cent., for twenty-four hours. Dehydrate as rapidly as possible and imbed in paraffin; cut sections as thin as possible; attach the sections to the slides with Meyer's albumin: wash out the paraffin with naphtha and alcohol; rinse the slide with distilled water; then cover the sections with distilled water and heat for ten minutes at 50° C. to 60° C. The top of the imbedding bath is a very good place for this. Pour off the water and cover with toludin blue, 1 in 4,000: replace in the paraffin bath for ten minutes; dehydrate clear and mount. Keep all the sections and, if you are lucky, some of them will be found to have the nerve fibres duly stained.

Some of the results obtained by this process are seen in the photo-micrographs which I now show (figs. 4 and 5). The first is taken from the incisor of a rabbit, the second from a transverse section, taken near the neck, of a human canine. But before I show you these I want to show what is the general result of the fixing in nitric acid, which you may consider a somewhat crude procedure, calculated to shrink and alter considerably the soft pulpal walls with which it comes into contact. Fig. 2 is a photo-micrograph from such a series, but stained with methylene instead of toludin blue. You will see that while the ground substance of the cells has been eaten away to some extent, very little, if any, appreciable shrinkage has occurred. This is better shown in the more highly magnified fig. 3; but before we leave this I would like to refresh

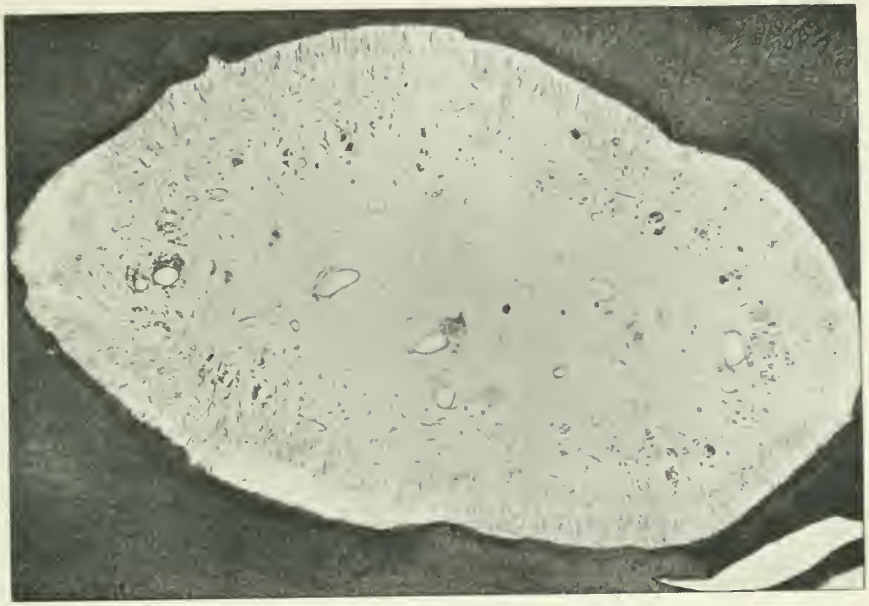


FIG. 2.
Human Canine. Transverse section at neck. Bethe methylene blue.

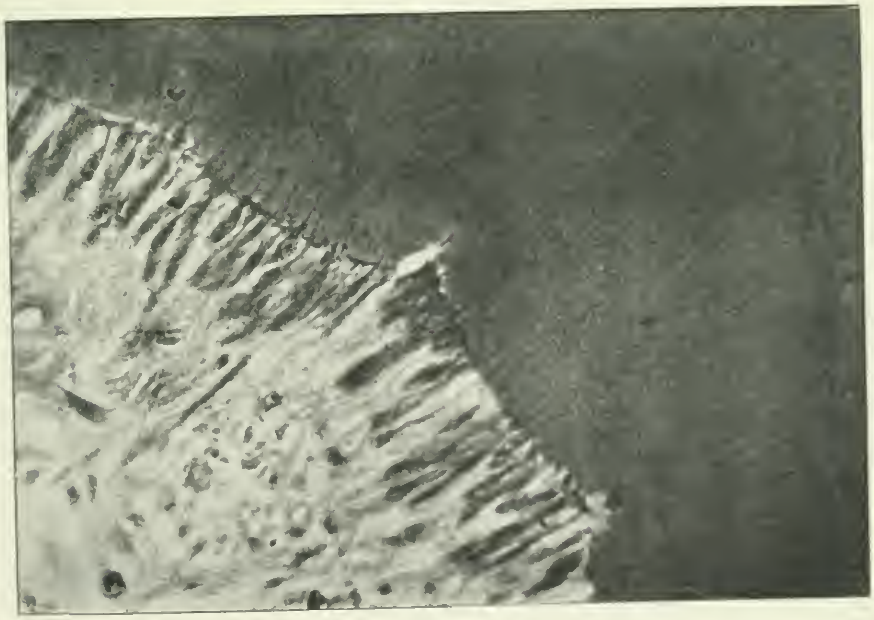


FIG. 3.
Same as fig. 2, but more highly magnified.

your memories as to the general distribution of the nerve bundles in this region of the pulp.

"Entering the apical foramina the bundles of medullated nerve fibres pass collectively into the pulp in lines directly corresponding to its long axis. In every case the chief nerve fasciculi run alongside the larger blood-vessels and in their areas of distribution follow them closely." Here you will see the blood-vessels running not in the centre of the pulp, but on the palatal and buccal aspects, and the main bundles of nerves running alongside them in the same sheaths. Arranged around the periphery of the pulp and just underneath the basal layer of Weil are the smaller bundles produced by the splitting up of these larger ones. The ultimate destination of these smaller bundles, then, is the problem to be solved. The next slide is from the same pulp more highly magnified and shows the odontoblast area, here two or three cells deep, with the odontoblast processes running into the dentinal tubules, and I think you will agree with me that it does not seem to have suffered much from its bath of nitric acid. Fig. 4 is from a transverse section of the incisor of a rabbit treated by the Bethe process and shows the odontoblast area again. Running up from the basal layer of Weil are a number of long nerve fibres which penetrate the odontoblasts right to the dentinal edge. These are still better seen in the next slide, taken from the same series of sections, but embracing also part of the dentine. These appearances led me to endeavour to stain a human pulp in the same way and the result is seen in fig. 5. This shows the same mass of nerve fibres running up from the basal layer, and you will see here one running directly from the medullated nerve bundle. It is to be noticed that these fibres are very much larger than one has been led to expect from early researches. Myriads of minute scopiform strands is the description by Röse, and other writers describe similar appearances of large numbers of interlacing fibres which have been called the plexus of Raschkow. In none of my sections stained by this or the Golgi process have I been able to obtain such appearances. On the contrary, I have found the nerve fibres to run directly, almost in straight lines, to their destination between the odontoblasts, and I am strongly of the opinion that the so-called plexus is not composed of nerve fibres at all, but is made up of connective tissue bundles running upwards and forming the structural skeleton of the pulp.

In none of my sections stained by the Bethe process, however, was I able to detect any passing of the nerve fibres into the dentinal tubules.

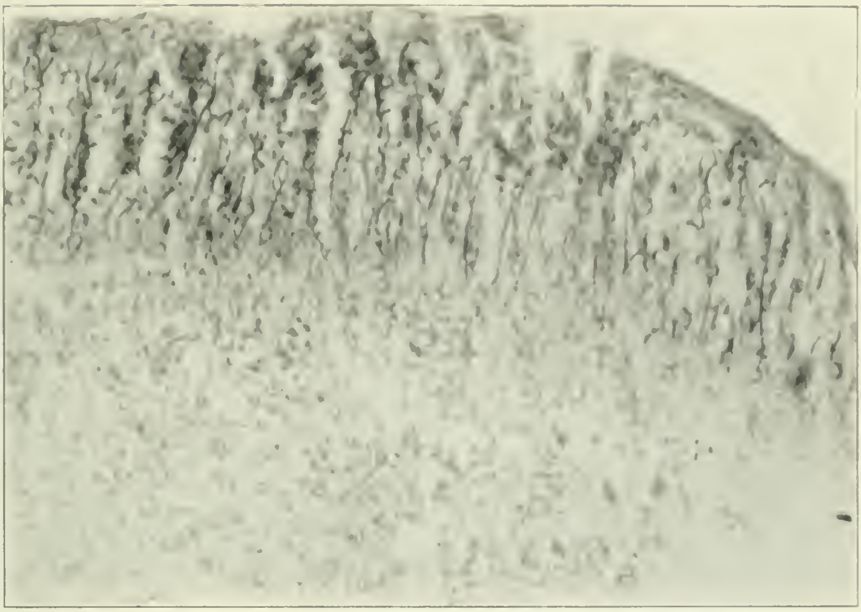


Fig. 4.
Rabbit Incisor. Bethe toludin blue. × 375.

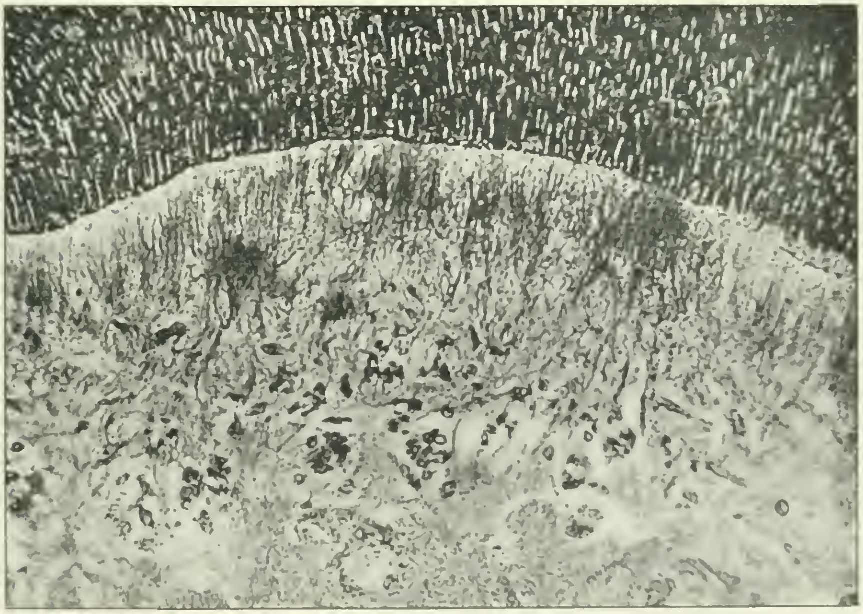


Fig. 5.
Human Canine. Transverse section at neck. Bethe toludin blue × 375.

The dentine in decalcified specimens stains in a very curious way, the exact significance of which I have not yet been able to make out. It was therefore necessary to try some other methods of staining, and Golgi's process was the next to give me any results. In spite of the suspicion cast upon this process as applied to teeth by the hasty conclusions of Morgenstern, and of the more recent methods of Ramon y Cajal, which are superseding it, Golgi's methods remain the classical methods of investigating the distribution of nerve fibres, and no worker can afford to ignore them. If used with due care and discrimination they give reliable results and offer certain advantages in the case of teeth, in that sections do not require to be very thin, but rather give better results when fairly thick. Golgi's rapid process was selected for use. Sound, freshly extracted teeth were sawn into small pieces or ground on a wheel so as to expose the pulps, and then immersed in a mixture of 8 c.c. of potassium bichromide, 2·5 per cent.; 2 c.c. of osmic acid, 1 per cent. The pieces were taken out in from three to five days and immersed in 75 per cent. silver nitrate for twenty-four hours, then embedded by the Koch-Weil process in hard balsam, and rubbed thin in the usual way with ground glass and pumice. This process seems to me the least open to objection of any process of obtaining sections of teeth with pulps in situ. No unusual or strong solutions of acids come in contact with the tissue, and compared with most of the other methods it is short and simple; its only drawback is the uncertainty of the results given by it. In some of them the appearances are due to tissues that have not as yet been described, whilst in others blood-vessels instead of nerves have been caught by the impregnation, but in successful specimens the appearances all point to the actual entry of the nerve fibres into the dentine. Fig. 6 is taken from a transverse section of a human canine just at its neck below the enamel. Here you see the black nerve fibres running into the dentine; from their position I think there can be no doubt of their nervous nature. Here the main nerve trunk round the blood-vessels is clearly seen, and these fibres run out from it in slightly sinuous fashion between the odontoblast cells, also slightly stained by the chromate of potash, and are clearly recognisable as such right up to and into the dentine itself.

Fig. 7 is taken from a longitudinal section of a human canine, again stained by Golgi's process. While the rest of the tissues are not shown as in the last specimens, and so the certainty of the identity of these with nerve fibres cannot be made out, a comparison with the toludin blue specimen shown earlier (fig. 5) will show that the two are strictly

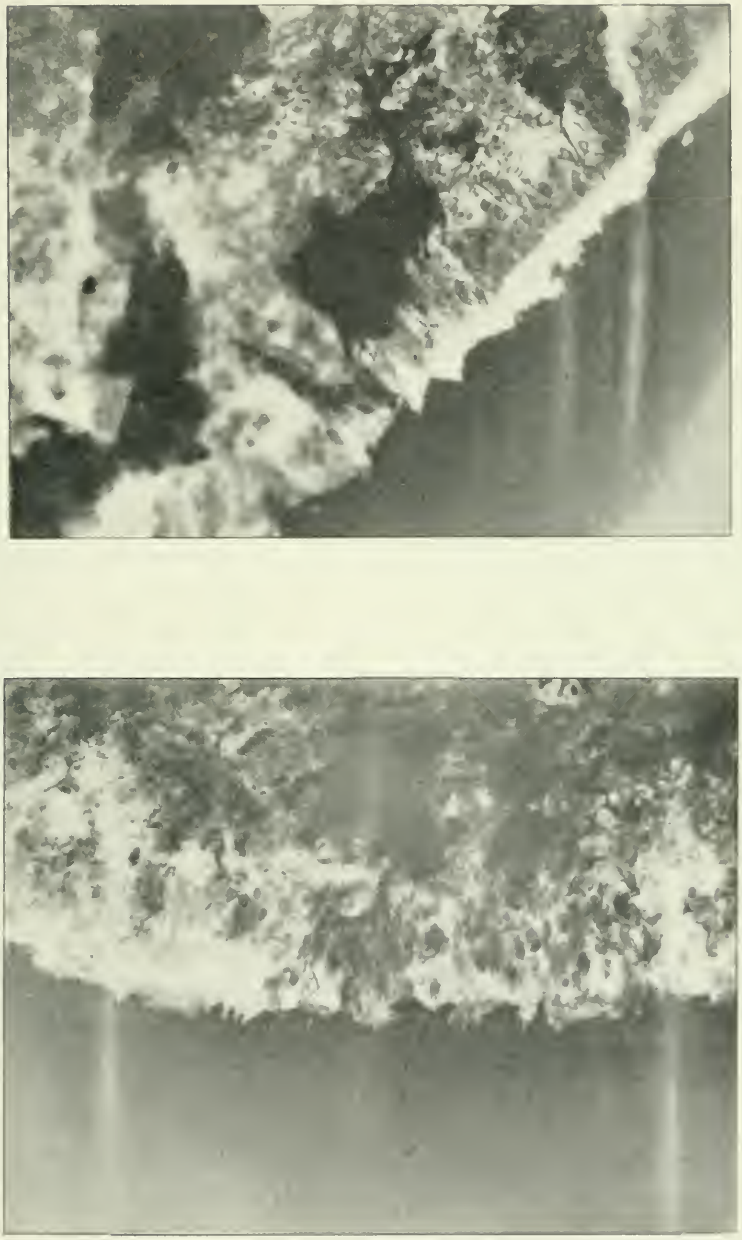


FIG. 6.

Human Canine. Transverse section. Golgi method. × 250.

in accord with one another, and this picture better than any other shows what I believe to be the true relation and destination of the non-medullated fibres of the pulp.

To sum up, these pieces of evidence seem to agree in the main with Römer's contention that there are nerve fibres in the dentine of mammalian teeth, and that the sensibility of dentine can thus be accounted for. With his statement that they diminish in number as the cervical edge of the tooth is approached, and that none are present in the roots, I am unable to agree, as, indeed, the clinical fact that the most sensitive dentine is that at or below the gum margin seems to indicate. Also I

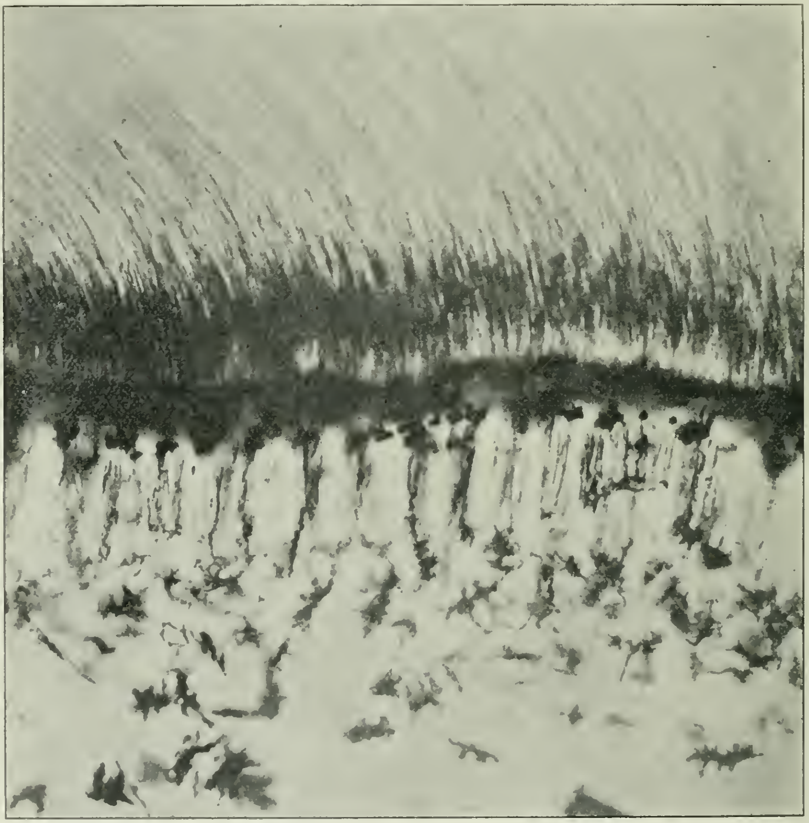


Fig. 7

Human Canine. Longitudinal section. Golgi method. 375.

have not as yet been able to obtain any appearance of these nerve fibres actually entering the odontoblast cells. In the second place I believe that the nerve fibres run directly to their destination and that the plexus of Raschkow, either as a true plexus or a mere interlacing of nerve fibres, does not exist at all, but that the appearances presented by it must be put down to some other cause.

I desire to tender my best thanks to Professor MacDonald, of the Sheffield University, who placed all the resources of his laboratory at my disposal, and without whose stimulating interest and help in every detail of the work this paper would never have been written.

DISCUSSION.

The PRESIDENT (Mr. Howard Mummery) expressed the indebtedness of the Section to the author for his investigation into a very difficult subject.

Mr. HOPEWELL-SMITH thought the Section was indebted to Mr. Law for bringing forward a paper on a most interesting subject. To himself the questions of the innervation of the pulp and the nutrition of the dentine were most fascinating, on account of the difficulties in connection with the working out of the various problems. He had had the pleasure of seeing Mr. Law's photographs a little while ago and knew of his work in Sheffield, and he thought it was a sign of the times that the younger members of the profession were investigating those matters. He took it that Mr. Law rather agreed with Oscar Römer's contention of the presence of nerves in the dentine. He himself had read Römer's paper when it was published some years ago (1899), and although there was a large number of drawings in that paper he thought Römer only gave two in which he showed some things which he called nerve fibres going into the dentinal tubes. If he had wanted to prove his point he should have drawn them a little more carefully and clearly shown the beads or gemmules of the non-medullated fibres, because that was the best way in which one could histologically discriminate between a strand of connective tissue fibre and a fibre of the non-medullate nervous system by the presence of little gemmules which were merely the nuclei of the neurolemma sheaths of the medullated fibre. But, as far as he remembered, Römer did not show any of these varicosities at all, and he might have been describing something quite different. Also Römer did not agree with the English idea in connection with the sheaths of Neumann. Römer confused the sheaths of the dentinal tubes with the dentinal processes of the odontoblasts, and made out that the odontoblasts were pierced by the non-medullated nerve fibres, the fibres passing up, in the substance of the dentinal fibrils, into the dentine. Römer thought there were no sheaths of Neumann at all, and therefore from that point of view it was very difficult to follow him. He should like to know whether, if it was true that there were nerves in dentine, there was any analogous condition elsewhere in the body. He knew of no nerves running in bony canals *per se*, and he did not see any reason why the dentinal tubules should contain nerves. He thought it would be a very bad thing if they did, as everyone would be afraid to take anything warm into the mouth owing to the terrible pain that would result from the presence of non-medullated fibres just beneath the enamel of the teeth. With regard to the plexus of Raschkow he was convinced that he had obtained it some years ago by the very simple experiment of fracturing a fresh tooth in a vice and removing a piece of the thin, colourless, serous exudation on the surface of the pulp and staining with 1 per cent. solution of gold chloride. He took it that it contained the interlacing fibres that had been described by Raschkow, close up, between the odontoblasts and the dentine. He still thought that Retzius and Huber were correct in considering that the non-medullated nerve fibres ended near the odontoblasts and did not penetrate the dentine. He (Mr. Hopewell-Smith) succeeded years ago in staining some non-medullated nerve fibres in the human pulp by adapting Dogiel's process to human teeth, and he thus demonstrated those extremely thin, delicate, beaded varicose fibres which histologists understood to be non-medullated nerve fibres. They were extremely minute and not easy to obtain. He used a $\frac{1}{16}$ per cent. solution of methylene blue in physiological salt solution, and fixed the stain by means of picrate of ammonia and mounted the pulps in glycerine. The result was that in a few fortunate preparations he could see the very thin beaded fibres, but he could not get them in situ, and that was the disappointing part about it. They were differentially stained with extreme difficulty, the process being stopped before the stain had had time to affect the other tissues. Mr. Law's paper would give rise to further thought, and no doubt stimulate work in connection with that very important subject.

Dr. E. I. SPRIGGS thought Mr. Law was to be congratulated on taking up the work, because there was, no doubt, a great deal still to be discovered in connection with the histology of the nerve tissues of the pulp, and that would only be found out by working with new methods and adapting old ones. He was quite sure that on such lines Mr. Law would obtain results of still greater value. Some years ago, when he himself had the duty of teaching the subject, he went to work in Römer's laboratory and saw the sections to which reference had been made that evening. Those sections certainly did show in some parts a fine fibril, stained with methylene blue, which was different, especially in size, from the ordinary dentinal process running into the tubule. Römer in his paper gave the measurements. The specimens only showed a few of these fibrils, and one had to be told where to look, and look carefully, before they could be seen. The fibrils, as far as he could remember, were about a third of the thickness of the odontoblastic process. The odontoblastic process, as everyone knew, had rather the appearance of a transparent band or tube; the fibrils in question looked solid, and very much resembled the processes seen in one of the specimens shown by Mr. Law, though Mr. Law's fibrils appeared a little thicker than those of Dr. Römer. He believed a combination of Dr. Römer's method of decalcification with the modern methods of cutting thin sections in celloidin should lead to an elucidation of the problem. Römer decalcified the specimens in formic acid without spoiling the capacity of the tissues for staining, and he believed this method was of great value. When nitric acid was used, except in very dilute alcoholic solutions, the tissues had the fatal appearance of acid-spoilt tissues: the nuclei stained imperfectly or not at all. Mr. Law's specimens showed this, but, fortunately, it did not affect nerve fibres so much as other cells. Römer's sections had the disadvantage of being rather thick. They were cut with a Yung microtome, and it was not possible to get them anything like so thin as they were required. He should like to suggest to Mr. Law to follow Römer's method of decalcification, and use a Delépine microtome to cut the sections. The method of decalcifying in 10 per cent. formic acid did not work unless the tissues had been hardened for a long time in formalin. When he himself was cutting sections of dental tissues, he found that specimens he had brought from Germany, that had been in formalin for a year or two, stained beautifully, and when cut thin showed all the proper nuclear staining, whereas those that had been in formalin for only a month did not. He thought the secret of decalcifying with formic acid was to keep the teeth a long time in formalin with the pulp chamber opened to get thorough fixation before exposing them to the action of the formic acid.

The PRESIDENT hoped to hear further results of Mr. Law's researches. It struck him that some of the fibres looked a little large and did not show any beading, and it would almost suggest that these might be connective tissue fibres. One of the great difficulties in searching for nerves in the dentine was that the sections could not be cut thin enough, and with thick sections it was exceedingly difficult to determine what one was looking at. That was the case with Morgenstern's specimens, which were so thick that satisfactory photographs could not be taken of them, and all the illustrations were drawn by hand.

Mr. W. J. LAW, in reply, said, with regard to thickness, that he had not made any measurements. Both the Golgi and the toludin blue methods were not staining methods pure and simple: the impregnation was not only in the fibres, but around even more than in the actual fibres themselves, and that was one reason why they seemed to be thick. He thought Dr. Spriggs had replied to Mr. Hopewell-Smith to a very large extent, and had said a great deal more than he could say about Römer's work, but he himself profoundly distrusted teasing methods. It was possible to obtain anything in that way, and there was no proof of the nature of the thing obtained except the little bit of beading Mr. Hopewell-Smith had described. He quite agreed that some of the specimens were rather thick, but thought the toludin blue specimens were as thin as they could be got, because not only was there a difficulty with the things themselves, but the dentine was very difficult to clear. He recommended anyone trying to cut sections of dentine in paraffin to use mineral naphtha as a clearing agent: it made the dentine far less brittle than any other agent he had used. The

Bethe specimens and the methylene blue specimens, which were 10 mils thick, were the thinnest he had been able to attain, and they were got by clearing in mineral naphtha. He had not used the formic acid process, not having been able to get the details. He felt his paper was only a start, but he thought that a review of the work done and the showing of a few results, about six sections in eighteen months, would perhaps tend to raise further interest in the matter.

The discussion on Mr. Colyer's paper on "The Treatment of Children from the Dental Aspect," adjourned from the previous meeting, was again postponed until the next meeting of the Section (March 23).

Odontological Section.

March 23, 1908.

Mr. J. Howard Mummery, President of the Section, in the Chair.

(1) Two Specimens of the Head and Jaws of the Adult Hemirhamphus.

(2) A Specimen showing Developmental Defects occurring in the Upper Jaw of a Pike (Esox lucius).

By A. Hopewell-Smith, M.R.C.S., L.D.S.

(1) The *Hemirhamphus* (*ἥμῐσυς ῥάμφος* = half-beak) belongs to the second subclass of fishes, the Teleostei, of which it forms one of the species of Physostomi. It attains the average length of about 2 ft. A fresh-water fish of tropical climates, it is viviparous. The specimens were displayed, one with the mouth open, the other with the mouth closed. The upper jaw, consisting of the intermaxillary bones only, is triangular in shape, slightly convex on its upper surface, and measures in its broadest diameter about 1·5 cm. The margin is beset with numerous small teeth. The lower jaw is prolonged into a beak about 7·5 cm. in length and consists of the dentary bone. The tooth-bearing portion of the beak, as also its greater portion, is osseous, but the extremity is cartilaginous or semi-cartilaginous. It is an example of the mandible in fishes undergoing adaptive modification, for the purpose of shovelling up into the mouth, from the sandy beds of the rivers, the small invertebrate creatures on which it subsists.

(2) A specimen of a remarkable example of developmental defect in the upper jaw of a pike (*Esox lucius*). The head—after division in a

sagittal direction—presented the left lateral half only. (The remaining portion of the specimen is to be found in the Museum of the Royal College of Surgeons of England [Catalogue No. 179A], having been presented to it by C. J. Seligmann, Esq.) The upper jaw falls short of the length of the lower jaw by about 4·5 cm. The bones affected are the intermaxillary, the maxillary, the palatine, the vomer, the pterygoid, and anterior frontal, which are not only shortened in their antero-posterior diameter, but are also bent downwards at a considerable angle with the lower jaw. There is great dilatation of the distal extremity of the maxillary bone. No traces of injury appear to exist; hence the assumption that the facial arrest has been occasioned by congenital circumstances.

Mr. Hopewell-Smith presented the specimens to the Museum of the Society.

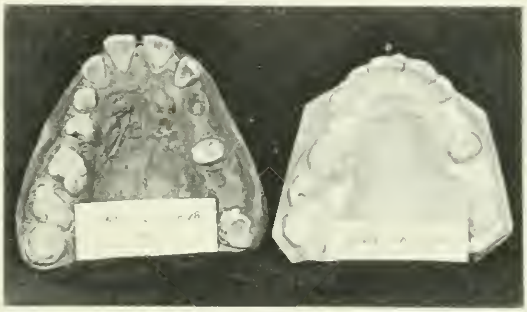
A Case demonstrating a Point in the Treatment of Pyorrhœa.

By Ernest Sturridge, L.D.S.

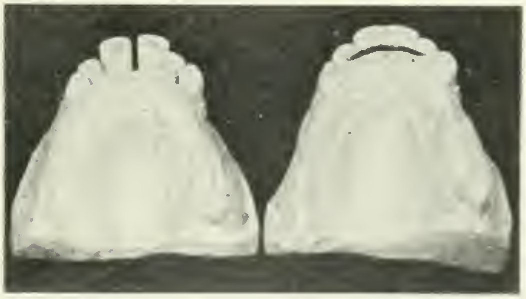
In bringing this case before you to-night, I desire to show a method I have adapted to overcome one of the knotty points in the treatment of pyorrhœa. We are all aware of that condition which arises in consequence of this disease by which teeth affected by pyorrhœa leave the normal position, protrude with interspaces, and become loose. In that abnormal position no amount of treatment will be productive of any permanent good results. On the other hand, if such teeth are replaced in normal apposition, *and retained there,* satisfactory results may be expected.

I have retained such cases by means of plates, with wire arches across the front teeth. For example, Case 1 (shown on screen): This case has been retained by a plate with a gold wire passing from the spaces where the bicuspids are missing in front of the incisors. The patient has been wearing this appliance, as a night plate, for the last ten years, and the slide shows the condition of the teeth as they now are. The pyorrhœa has never recurred in any of the incisors, which were originally badly affected by the disease and loose. But this form of retention has its disadvantages, in so much as that the wire retention arch is likely to get out of position or get broken.

The method I propose to bring to your notice now seems to me to be more satisfactory, in that the teeth are held perfectly rigid, and are therefore functionally more useful; besides, it does away with the necessity for a plate, which is an advantage where no teeth are missing.



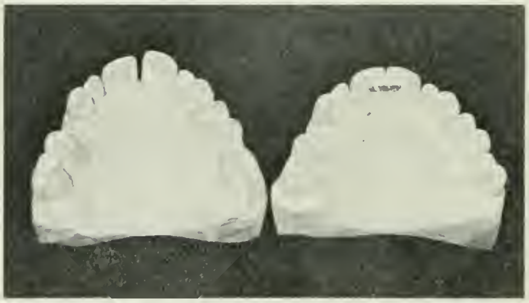
Case 1.



Case 2.

I will explain the method briefly (Case 2.) The teeth protruded in the incisor region in the manner shown, the incisors being very loose, with pyorrhœal discharge from the palatal and mesial aspects. The deposits were removed, and at subsequent sittings the diseased roots treated by cataphoresis. The incisors were brought into the position they now are seen in by an Angle's wire traction arch and bar, with pressure in the usual way. When in position (which took about ten days) a drill hole, the diameter of a wire the size of No. A wire gauge, was made on the palatal surface of each incisor, midway between pulp and approximal surface, extended some distance into the tooth in the direction of the root, and parallel to each other; into these drill holes were placed platinum pins a trifle smaller than the holes made, the heads of the pins extending a little distance out. An impression of this surface of the teeth was taken and the pins removed with it. A plaster and sand model was made, which, when separated, retained the pins in their relative proper positions; platinum foil was then burnished from

Case 3.



Case 4.

pin to pin, and on this, also from pin to pin, was soldered stiff plate wire strengtheners, making when completed a rigid wire-like bar holding the pins. This was finished down as small as consistent with strength of the appliance, and when completed it was cemented into position. It holds the teeth perfectly rigid, as many of you have seen to-night.

Case 3 shows three teeth held together in a similar manner. Pyorrhœa existed between the central and lateral, the central being twisted on its axis and much out of position. This case was specially adapted for a staple, the patient being a young lady who did not want to wear a plate. The central was brought into line by my colleague, Mr. Lockett, and stapled by me in the manner shown.

Case 4, as you see, is one in which the central on one side was affected at the mesial aspect; the case has been done some years and is now in a perfectly healthy condition.

Case 5, an X-ray, shows a staple placed in the cutting edge of two centrals, a very easy and effective position to construct a staple when the cutting edge has worn down a bit. This particular case was almost hopeless when attempted and eventually failed, but others I have done in similar positions have succeeded admirably.

The point I wish to bring forward is, that in the treatment of pyorrhœa, teeth which have taken an abnormal position should be replaced in the normal, and, if retained there, will make a good recovery, but if no attempt is made to replace and retain such teeth, treatment will avail little.

DISCUSSION.

Mr. ROBBINS thought Mr. Sturridge had made a step in advance. He himself had never been so successful in the treatment of pyorrhœa as he had hoped at first; he had managed to keep the disease under, but was never able to report an absolute cure of an extreme case. He was inclined to think it was apparently because he had not stapled the teeth in some way or other. The extent to which he had gone had been to fasten the teeth by very fine gold wire bindings. In one patient, under treatment for some years, he was able to keep the disease down, but could not cure it, and he passed the case on to Mr. Sturridge to treat with cataphoresis; he saw the patient six months after the cure was effected, and her mouth was in a perfect condition.

Mr. BADCOCK felt indebted to Mr. Sturridge, not only for the very useful things he had said that evening, but because under Mr. Sturridge's influence he himself some time ago started treating cases of pyorrhœa by cataphoresis, and his success had been very considerable. There was not yet sufficient time to say whether any absolute cures could be claimed, but so far as he could judge it was the most successful method of treatment he had ever employed for pyorrhœa. In many cases the pyorrhœa was treated before there had been any movement of the teeth at all, and in those cases it was fairly easy to check suppuration. Whether it would recur or not his own experience of the method was not sufficiently long to say. The question was, what became of the pockets? His own belief was that once a pocket had formed it always remained, unless it was destroyed by the removal or the natural shrinking of the gum. Although when treating cases electrically the gum round the tooth tightened up to a remarkable degree, and appeared perfectly healthy and firmly attached to the tooth, a pocket remained even though there might be no suppuration. He should like to know whether any member had ever known a pocket to be obliterated by the reattachment of the gum to the tooth.

Mr. W. J. MAY had held the same view, that where a pocket had been long in existence it could not be obliterated. To a casual observer the pocket was invisible, yet on careful examination a fine bristle could be passed some distance up the side of the tooth. He had found the splints Mr. Sturridge had referred to of particular value. His first case was one in which a patient came to have a loose tooth removed. The pulp was alive, and the trouble was mainly due to the bite. He eased the bite, painted with iodine, and the tooth got better. A year after the patient came back again, thinking he had better have the tooth out as it had suddenly developed periostitis. The same method was adopted, but a year later there was no other method of saving it except by placing a splint. The patient was unable to bite squarely on the top of the tooth, and the tooth shifted in its socket every time the patient bit. It was a molar tooth, and he placed a small splint, with a pin in the crown of the molar and a short pin in the crown of the bicuspid, and fixed the two together with a small piece of wire, making a very slight biting surface in order to give a certain amount of utility. Within quite a short time it became fixed, and the amount of pressure that could be exerted was as much as one could exert with fingers and thumb, and caused no pain. The first treatment of that tooth began seven years ago. Lately the splint had broken, and he discovered the tooth was still loose, but at any rate tighter than it was five years ago. The splint had been replaced and there was still a powerful bite. He had used the method in several cases, and had met with a great deal of success, although naturally there were certain failures. Another instance where the splint was useful was where the first molar had been lost and the second molar, having tilted forward and become somewhat loose, had caused food to collect between it and the third molar, with consequent decay. In these cases a small hole could be drilled in the crown of each molar and the two fixed together by means of a piece of plate with a pin fitting each hole. The fixing of the teeth gave mutual support, and prevented the collection of food.

Dr. KIRK DAVENPORT thought the abscess pocket was being treated with far too much respect. His own practice for three or four years was to remove all deposits and then to massage the gums carefully, and in the bad cases, where the pockets were deep and there was a large amount of pus discharging, he deliberately crushed the alveolar plate in. His method was to take a pair of pliers, and with a bit of amadou dipped in antiseptic deliberately to crush in the alveolus, forcing the entire bone tight round the teeth. That allowed the process to come in contact with a tooth, and gave an opportunity for the deposition of bone cells, which, he felt certain, took place. He had no positive proof of that, but after a year or two he had found it impossible to pass a bristle round the necks of the teeth. He thought everyone would gain by using Mr. Sturridge's method of fastening the teeth together.

Mr. W. HERN also thanked Mr. Sturridge for showing the devices by which he steadied teeth. He did not think the method was a cure for pyorrhœa, but the pyorrhœa could be treated at the same time. With regard to one of the cases shown by Mr. Sturridge, in which a left central had moved forward and was affected with pyorrhœa, it was interesting to hear that the case had been regulated early in life. He himself had seen several such cases in which pyorrhœa had affected a central incisor or two central incisors later in life after regulation had been carried out. He should like to know whether a regulation device had been worn on any of the teeth of Mr. Sturridge's other cases that had subsequently needed the steadying device. He believed that pyorrhœa was much more likely to affect a tooth in after life when it had been moved mechanically in early life.

Mr. H. BALDWIN said in quite a number of cases where teeth had been very much moved forward out of position by pyorrhœa, he had treated the cases as ordinary regulation cases and had never had to regret having done so. On the average in such cases the teeth had lasted perhaps ten years longer than they otherwise would have done. He had never actually splinted together; he generally used a plate and wire springs round the front to pull the teeth back into position, the plate being always worn at night by the patient as a retention plate. The mere fact of pulling the teeth into position certainly seemed to help in the cure or half-cure of the pyorrhœa. Of course the pyorrhœa had also been treated locally by antiseptics, scaling, and so on.

Mr. STURRIDGE, in reply, was very pleased to hear what Mr. Badcock had said about the treatment of pyorrhœa by cataphoresis. Mr. Badcock was one who had taken it up on his advice, and therefore he was glad to hear he had had such good results. With regard to pockets, he believed in cases where the teeth had moved out of their positions, if they were brought a little further back than the normal, so that pressure was brought to bear on the root by the alveolus, the pockets were practically obliterated, but there was no reattachment: a very fine bristle could often go to the bottom of the original pocket, which remained perfectly healthy. In the first case he had shown, he believed it was possible to get a bristle into the pocket. Provided the deposit was thoroughly removed the teeth might be kept perfectly healthy and firm. He himself had always used the treatment for nothing but pyorrhœa, but it was a good idea of Mr. May's to staple molars with troublesome interspaces into which food might lodge and thus save a lot of trouble. One of the commonest causes of pyorrhœa was the regulation of the teeth in early life, but he thought it depended a great deal on the way it was done. In two cases he had shown that evening—the one with three teeth stapled together and the one with two teeth—the disease was undoubtedly caused by regulation. With regard to Dr. Davenport's remarks about crushing the alveolus, that was carrying out his own idea in the opposite direction; Dr. Davenport pressed the alveolus towards the tooth, while he pressed the tooth towards the alveolus. It was a matter of getting the alveolus close up to the teeth, removing all the deposit, and treating the cases.

The Treatment of Children from the Dental Aspect.

[*Discussion on Mr. J. F. Colyer's Paper, read January* 27, 1908 (*see p.* 33).]

Mr. WILLIAM HERN said the paper was a valuable and important one, as it dealt with those who were to be the men and women of the future. Mr. Colyer had done him the honour of referring to a remark he had made some time ago at the meeting of the Metropolitan Branch of the British Dental Association, when he said the work of Miller was an epoch-making one, inasmuch as it showed that the main cause of decay was the lodgment and fermentation of carbohydrate food on and about the teeth. He was afraid he had still to admit that in his view the main causes of dental caries were unknown. The facts were that caries did not affect all parts of a tooth equally; that it commenced in fissures and pits or on the interstitial surfaces where food might lodge, whereas the cusps and cutting edges of teeth were practically exempt from the disease; that the convex surfaces of teeth, which were swept by the movements of the lips, the tongue, and the food, were exceedingly seldom the starting place of caries, except in patients in whom these natural cleansing processes were hindered by means of plates or mechanical devices. Further, it was noticed that there was a progressive diminution in the amount of caries in patients in proportion to the amount of intelligent care they bestowed in looking after the teeth. The terrible amount of caries of the neglected teeth of hospital patients was well known. The condition, too, of the mouths of private patients varied so much in proportion to the care taken by them that he could divide them into about five classes: the badly kept, moderately kept, fairly well kept, well kept, very well kept. Professor Miller's classical experiments on the production of caries out of the mouth appeared to him to clinch the whole matter. Professor Miller showed that caries could be produced out of the mouth, and he demonstrated conclusively that the cause of caries was the fermentation of carbohydrate foodstuffs. It was not denied that certain other conditions might have some subsidiary action in producing caries, but they only served to emphasize that statement and contention; for instance, Mr. Colyer brought forward a long list, such as the changed character of the food, and quoted Dr. Sim Wallace with regard to the ridding of the foodstuffs of their fibrous parts by methods of preparation, thus rendering them more liable to lodge about the teeth. Mr. Colyer also spoke of the increased fermentability of the carbohydrates and the introduction of monosaccharides, and mentioned the capillary action of acid from one carbohydrate, viz., sugar between the teeth. He did not think Mr. Colyer quite gave Professor Miller credit for his views on the fermentation of sugar, because Miller did not overlook the fact that sugar was very fermentable and produced acid; he said that starch produced acid rather more quickly and more copiously than sugar. Then Mr. Colyer spoke of the roller milled flour being more acid-producing, and that might be so if it was rendered more agglutinative, but the chemical constituents of flour were the same now as they always were, and so also were those of potatoes and rice and of many other carbohydrate foods. With regard to the varying resistance of teeth, and the varying physical character of the salts entering into their composition, he thought everyone would agree with Mr. Colyer that this was a factor influencing caries. They all probably recognised types of teeth very prone to break down, and they were called weak teeth; in other mouths were teeth of quite a different character, and they were called strong teeth. But it should be remembered that, whether in the strong tooth or in the weak one, the decay, when it occurred, was in the same position, viz., in the pit or valley, or interstitially, and therefore he must still maintain that the chief cause of decay was fermentation of the carbohydrate foodstuffs. He agreed with Mr. Colyer that attention should be concentrated on the prevention of the disease, but could not go quite with him in regard to the main lines of preventive treatment. Inasmuch as carbohydrate foodstuffs could not be avoided, they must not be allowed to rest and ferment on the teeth. Observation showed that decay did not take place in certain positions on the teeth; therefore, Nature's method of prevention by constant movement and friction should be adopted in treatment. With regard to preventive treatment, Mr. Colyer mentioned the insistence of breast feeding, the use in early years of foodstuffs which required efficient mastication, the insistence of mastication by the child, and the use of carbohydrates, which are not easily fermentable, and, secondary in importance, the proper use of the toothbrush. Although good in themselves, he himself should reverse those suggestions, and say that the first line of defence was the toothbrush, systematically and intelligently used. The first thing to do was to draw patients' attention to the main facts about decay and to the importance of mechanical cleansers. A tooth-pick or floss silk was almost as important as the toothbrush for this purpose. The toothbrush should be stiff and small, and patients should be taught not only to brush their teeth, but their gums as well. A large amount of the pyorrhœa of the present day was due, in his opinion, to the want of friction on the gums. Mr. Colyer rightly pointed to the fact that patients who were mouth breathers had gingivitis about their front teeth, whereas the gums at the back of the mouth were healthy; these conditions were due, no doubt, to the fact that many of the cases were protrusion cases, and there was little or no friction by mastication on the gum about the front teeth, whilst the gums about the back teeth of these patients were frictionised by mastication. Mr. Colyer said: "With regard to curative treatment we must always keep clearly in mind the necessity of rendering the mouth functional." If by that Mr. Colyer meant rendering the teeth functional he quite agreed with him; but Mr. Colyer was a little paradoxical, because further down he said: "By extraction we are more likely to ensure the mouth being rendered functional." Personally, he himself was very keen on saving the temporary teeth. A child was a growing and a developing animal, and if that child could not masticate properly it could not develop as it should. The size of the second temporary molars, too, showed the physiological importance which Nature placed upon their action; they were nearly as large as a permanent tooth, and he thought the retention of all second temporary molars at any rate was very important. There must be exceptional cases, in hospital practice especially, where patients came with a tooth carious, and perhaps suppurating, when extraction might be necessary, but he could not agree with Mr. Colyer in the somewhat lavish way in which he extracted these teeth. In Messrs. Smale and Colyer's "Diseases and Injuries of Teeth," they state "that temporary teeth should be kept as long as possible, for by this means we best ensure the growth of the jaw, and prevent many forms of irregularity that one meets with in permanent teeth," and also: "The second temporary molar should not be extracted until the permanent successor is ready to be erupted. On this point too much stress cannot be laid, and even when an alveolar abscess arises it is better to open it within the mouth than to remove the tooth." Personally he was in agreement with those lines of treatment, and he much preferred Mr. Colyer's old method of treatment to his new, for he thought that the rather liberal extraction suggested in the paper was open to serious objection.

Mr. C. Robbins said the paper was a valuable one and bristled with debatable points, but he wished to confine his attention only to one point, and that was the sweets bogy, which, he thought, had been for ever laid to rest by Dr. Miller. Mr. Colyer was of opinion that the cause of decay was unknown, and then set about to trace it largely to the use of sweetstuffs, believing that the harm done to the human race by constant eating of sweetstuffs ran alcohol very close. Indeed, Mr Colyer became bolder as he proceeded, and went so far as to say that sweets in any form of sweetmeats should be forbidden. Now, if all that were true, and not simply the honest opinion of one man, then he personally ought that evening to be sitting in sackcloth and ashes bemoaning the wrongdoing of more than a quarter of a century. When he started in practice in the early eighties he was steeped in text-book orthodoxy, but contact with human nature by the chair side and a riper experience brought about an alteration, and after a time he felt bold enough to think for himself. There was always one matter which from the first bulked largely in his professional ambition, and that was to win over the confidence and even the affection of the very youngest of his patients, in order that he might be the more useful to them at that and subsequent stages. He considered it a sacred trust to win the affections of the little ones, so that the dentist might be regarded as the friend of the family. In dealing with children he never took them in the least by surprise, was always strictly straight and honest with them, and let them see that it always hurt him to hurt them, and in that way a nice feeling was generated between the operator and the patient. For the children who behaved well he had kept a stock of pretty coloured little boxes, each of which contained a small number of pure sweets. Parents would often say: "I thought sweets were the things that did all the mischief," and that gave him an opportunity of explaining that a sweet or two in the daytime, when the saliva was being constantly changed, and when the teeth were well kept, never did a child any harm, unless for medical reasons sugar was contraindicated; and it also gave an opportunity of teaching the parent that the only danger was to give a sweet to the child when going to bed, because the sugar was not washed away by the saliva during the night, and, remaining around the teeth, produced a weak acid before morning; it was that acid which did the mischief. It also afforded an opportunity of emphasising the fact that the last thing to do before going to bed was thoroughly to clean the teeth. His child patients grew up, married, and brought him the second generation, who came absolutely without fear or dread; indeed, they looked forward to coming. During all that period he had watched carefully for any harm arising from his method and had failed to find any, and he meant to continue the practice right to the end of his professional career, unless he had some stronger proofs and better arguments than the paper afforded that a sweet or two in the daytime ever hurt a child's teeth. He was perfectly in accord with Mr. Colyer when he said that "it was a fatal mistake to jump at conclusions."

Mr. C. N. PEACOCK said, in advising his patients as to how to preserve their teeth, the first requirement for the teeth was hard work and the second was cleanliness. White bread, he found, did not give the teeth hard work. Some three or four years ago he started living on wholemeal bread, but found that it had not sufficient nourishment in it, and came to the conclusion that to get the best results one must use white bread toasted. He believed that the roller mill white flour was a much greater cause of decay of the teeth than sugar by reason of its finely powdered condition.

Mr. W. RUSHTON said there was so much in the paper with which he was in accord that his adverse criticism could not be very great. It was a great pity that Mr. Colyer did not divide his paper into the treatment of the teeth of the poor and the treatment of the teeth of well-to-do children, because the two things at the present time were so vitally separated. He thought the treatment of the latter was pretty straightforward sort of work: the parents, as a rule, were intelligent and the treatment prescribed was carried out. The conditions were different with the children seen in hospital practice. It was almost appalling to know what to do with the teeth of the children of the poor, not only in great cities but in rural districts as well, where the children's teeth were in just as deplorable a condition as they were in the large towns. The point brought forward with regard to glucose was very important. It was a well-known fact that the negroes on the sugar cane plantations had excellent teeth, although they were always sucking sugar cane, and he had little doubt that sugar cane did not do much harm. Boys at school no doubt harmed themselves by sucking sweets, but decay was found in boys who lived at home with very small opportunity for sucking sweets and who cleaned their teeth regularly. He agreed with Mr. Colyer that the essential causes of dental caries were not known. Exceptions could be found to every explanation brought forward. He thought roller milled bread and biscuit flour had probably a very great deal to do with it, and it was more than probable it might also be caused by the absence of fluorides and other salts that had been eliminated from the flour. The whole matter required careful examination, and he did not think chemistry alone would do it. Mr. Hern said that the chemistry of the thing was the same, but, as Mr. Colyer pointed out, it was not only the chemistry but the physical disposition of the chemical products that made all the difference between a hard tooth or a soft tooth. He had formed the opinion that children could have quite as good teeth when reared by artificial means as children breast-fed, but he thought it very much depended on how they were artificially fed. Much of the wrongful feeding of children at the present day was open to very great reform and might be made a Government matter with advantage. It was extraordinary how often physicians and nurses, more particularly the nurses, discouraged the mother from nursing her own child—very often with the idea, probably, of proving how remarkably clever the nurse was in substituting artificial means. He strongly believed in teaching the child to masticate hard food and in preventing it getting into the habit of washing down its food with liquids. With regard to sweetmeats, he agreed with Mr. Robbins that in the course of the day they did little or no harm, but the pernicious habit of giving a child sweets the last thing at night should be condemned. He thought the toothbrush still had its use, but in many cases it was a very overrated thing, even when it was properly used. He agreed with Mr. Hern that the toothbrush was not so important a cleansing agent as the tooth-pick or dental silk. He had been surprised more than once by the mother of a child, whose temporary teeth had decayed, saying to him that Mr. So-and-so did not fill children's teeth. It seemed to him a very terrible indictment against the profession that there should be practitioners who refused to fill the teeth of children whose teeth were in a carious condition. He disagreed with Mr. Hern that caries of the teeth occurred in inverse ratio to intelligent care. Some of the most careful and intelligent patients had teeth that in spite of every care decayed in the most terrible manner, while in hospital practice there were patients to whom the toothbrush was unknown and the teeth seemed to suffer in nothing like a corresponding proportion. He agreed with Mr. Colyer that the whole subject required more attention than had hitherto been given to it.

Mr. F. J. BENNETT thought there could hardly be a greater confirmation of Mr. Colyer's remarks as to the ambiguity which existed with regard to decay than the observations of the first and second speakers that evening. One started by assuming that decay of the teeth was due to carbohydrate action and the other said that carbohydrates in the form of sweets were the proper things to give to children. There could be no question that much was known about caries, but the final and intimate facts connected with it were still unknown. Not a word had been said that evening about micro-organisms, although Dr. Miller gave a very considerable amount of attention to them. He congratulated Mr. Colyer on speaking out plainly what he thought, and, believing that it was important to meet the matter in something more than a conversational way, he had ventured to prepare a resolution which he submitted to the meeting: "That a subcommittee be appointed to consider and report upon the best method of arriving at an exact knowledge of the causes of dental caries." The scope of this committee would be to review the methods hitherto adopted, and if necessary to suggest new lines of inquiry in keeping with the advances of modern science. He did not propose that personal investigation into the causes of caries should take place, but that the committee should review the methods adopted and consider modern methods and report to the Section. The subject had been discussed throughout the whole life of the late Odontological Society, and he thought the time had come when, as a branch of the Royal Society of Medicine, serious attention should be given to the matter.

Mr. MARTIN HENRY seconded the motion, believing that the whole subject of the paper resolved itself into a question of the cause of caries. Mr. Hern had spoken of the liability of some parts of the teeth more than others to decay, and it was obvious that if a tooth was weak a fissure was more liable to decay than a smoother part; it really depended on the condition of the substance of the tooth. With regard to Mr. Robbins and his sweets, twenty-five years ago sweets might not have done teeth such harm as they did to-day. The question was how long the terrible liability to caries had existed, and that was a matter worth inquiring into.

Dr. SIM WALLACE said there seemed to be no unanimity of opinion that it was possible to bring up children without a speck of decay in their teeth. Yet it was easily possible. There was only one way to prevent children fearing the dentist, and that was to bring them up in such a manner that they never required to have a single decayed tooth filled. It was his conviction that it was absolutely unnecessary for decay to appear in any children's teeth if they were brought up physiologically from the beginning and given hard food at an early age as recommended in Mr. Colyer's paper. He exhibited a model of a child, aged 7, who as far as heredity was concerned should have been expected to have had extremely bad teeth. The child was brought up on the bottle and had practically never used a toothbrush. The teeth were perfectly clean, and when he showed the child to a colleague he asked why a toothbrush should be used as there was really no necessity for it. Surely if the brushing of teeth were to have a marked beneficial effect, as is supposed by some, the amount of caries at the present day should be far *less* than it was fifty years ago! With regard to the preventive treatment spoken of, he agreed with Mr. Colyer in the necessity of breast feeding, but the trouble was chiefly with having it carried out. With regard to food requiring mastication, there was a fashion amongst medical men to advocate a pappy system of feeding, a system that was based on pure theory that initially was right, but in its development was wrong. The theory was that milk was the ideal food for children—no doubt it was for *infants* without teeth; but progressively it became a less ideal food until the child had twenty teeth, when it was not an ideal food at all. If a more or less exclusively milk diet was continued, then it would be found the teeth would decay. Medical men found poor, miserable specimens of children who had been very improperly fed, and who in all probability had not the power of masticating. They prescribed to them a diet consisting largely of milk, and those children, of course, thrived upon it. On the other hand, they found a great deal of harm done by milk. They found that cows' milk formed large clots in the stomach and gave rise to various intestinal disorders which they set about to try and prevent. In order to effect this they said the curd must be broken up, and in order to break the curd they recommended bread well soaked in milk, and the result had been a pernicious system of pap feeding. This was all unphysiological; the teeth were there to be used and the child could be given at a very early age a crust or piece of toast to exercise its teeth upon. When they had a complete set of twenty teeth there was no reason for giving them soft food at all, but there was no need to limit them exclusively to hard food; they could then eat practically any wholesome food they liked. The dietary of a child was unnecessarily restricted at the present day and consisted very largely of bread soaked in milk, milk puddings and porridge and milk, with occasionally potatoes soaked in gravy. With reference to the insistence on mastication by the child, that was included in the previous proposition, because it was impossible to teach a child to masticate if at the same time it were brought up on the milk and milk-soaked foods he had spoken of. With regard to Mr. Colyer's last point, the use of carbohydrates, which were not easily fermentable, and forbidding sweetmeats, that was perhaps a little sweeping. He had no objection to any foods that had come into general use; all such foods might be used, and at the end the child would have no dental caries whatever if proper precautions had been taken. A system he advocated was one that might be considered only for the rich, namely, that when sweets were consumed the sweets should be followed by dessert. Bread formed a very large proportion of the food of the poor, and bread contained an excess of carbohydrates, and there was therefore no need whatever of supplementing that food with sweets; what the poor should spend more money upon was albumen and fat. It had been said that bread was not an ideal food, but it was as near the ideal as any one single food. It stimulated mastication sufficiently for the child to feel instinctively that it it had consumed a considerable amount of starch by the time it had eaten a slice of bread, and as a consequence the child had no craving for sweets. The craving for sweets was largely a pathological craving brought about by pap feeding. With bread soaked in milk the child passed quantities of carbohydrates rapidly down the throat and the palate was cheated ; it did not appreciate the amount of carbohydrates which had been consumed, and consequently it craved for that very thing out of which it had been cheated, namely, starch and converted starch or its equivalent, sugar. Children brought up on hard food did not have anything like the same craving for sweets that those brought up on what he might call the "cheatery" system had. To give an illustration, the boy whose model he had shown, by way of diversion between courses, ate pats of butter, and there was no kind of fat that he would not take willingly and alone. Medical men recognised that fat was very valuable for children. With regard to the rich, he would not like to prevent them altogether from having sweets in the way of bonbons, jams, marmalade, cake, and so on : but when those things were consumed it was necessary that the mouth should be kept clean. This could easily be done in the manner already indicated. A simple experiment was to take a little chocolate and look into the child's mouth when it was supposed to have finished it. The chocolate would be seen in the crevices of the teeth. Then, if the child was given a piece of apple and its mouth examined immediately afterwards it would be seen that all the chocolate was gone and the mouth was clean. Consequently the easy way of getting over the difficulty of sweets leaving a dirty coating about the teeth was to allow them to masticate some fresh fruit afterwards. Fruit was not only slightly acid and stimulated the saliva, but in all probability had a tendency to discourage the growth of those micro-organisms which tended to decay the teeth. Mr. Colyer gave the percentage of children that would be freed from dental disease if the régime we advocate were pursued, and he (Dr. Wallace) believed it was essentially correct from an analysis of ten children he had under his care brought up on the method advocated. The ten children were aged between 5 and 7 and they had no decayed teeth whatever, whereas in a corresponding number of children of the same age one would expect to have in all about ninety decayed teeth. A little further on Mr. Colyer referred to another disease and the necessity of proper nasal breathing. He himself thought 20 to 25 per cent. of dental diseases could be put down to adenoids or mouth breathing, and he believed it was possible to find out why adenoids were very prevalent at the present day. With regard to the curative treatment mentioned by Mr. Colyer, theoretically it was correct, and in hospital practice, at least, the extreme measures were perhaps justifiable. Above all, one must keep clearly in mind the necessity of rendering the mouth functional, because unless that was done no amount of tooth brushing would keep the mouth clean.

Mr. STANLEY MUMMERY thought Professor Miller had proved the pathology of caries, and it remained to discover its etiology, and he did not think that the question of food or pits in the teeth or sugar, or any of those things, went deep enough. The real cause of decay he believed to be the susceptibility of different teeth. Practically speaking, savage man had no dental caries, and the reason was not very far to seek. An individual of a savage tribe developing dental caries would, having regard to the nature of his food, be weakened in the struggle for existence, and natural selection would very soon wipe him out altogether, and in that way a savage race became immune to dental caries. A civilised race fed on soft food, and could get on just as well with half its teeth as with the full number. The unfit could survive, and it was not a matter for surprise that dental caries was on the increase. It was the same with other diseases.

Mr. STANLEY COLYER, referring to this question of sugar, said he was one who believed that sugar was the chief cause of caries. He would like to correct one wrong impression, namely, that the teeth of workers on sugar plantations never decayed. In a paper by Ricordi on the teeth of coolies on the plantations in Natal, the writer stated that all the coolies when first engaged were warned against chewing the sugar cane, but for the most part they disregarded this advice, and within two years their teeth were practically all decayed. If Ricordi's facts were correct, then they seemed to show that the teeth of workers on sugar plantations might decay.

Mr. STURRIDGE could not agree with Mr. Stanley Colyer, because he had seen a great deal of sugar plantations in his life, and the East Indian coolies have the most beautiful sets of teeth he had ever seen. Probably the sugar might affect the teeth, but the sugar cane was a fibre, and when chewed made a most perfect toothbrush. He himself had eaten sugar cane during the whole of his younger life, and he knew a lot of other children who chewed sugar cane, and their teeth were really very good. It was the same with the negroes working on the sugar estates : many of them had magnificent teeth. The change came when they went into the towns and worked in the cities, and the teeth were not brushed with the sugar cane. When the East Indians left the sugar estates and went into the towns and cities they had a craving still to continue the chewing, and they chewed a fibrous stick called chew stick, which made a very effective brush.

Mr. BALY said that unless it was known what other diet the natives used he did not think it could be said that caries was due to the sugar alone. In Cape Colony the native Kaffirs fed on their native food had excellent teeth, but when a native servant was engaged he would not go as a domestic servant unless it was agreed to give him white bread, with the result that the Kaffirs working in the towns had very bad teeth, whereas those in the Kaffir locations, where they chiefly fed on mealies, had very good teeth.

Mr. J. F. COLYER, in reply, said the discussion had shown him quite clearly the chaos that existed in the dental profession with regard to the etiology of caries. It was apparent to him that there was considerable difference of opinion as to the meaning of the words "etiology" and "pathology." He respected Dr. Miller's work as much as anyone, but he still maintained that with the publication of that work on the micro-organisms of the mouth it was thought that dentistry had found the cause of decay, when dentistry had done nothing of the kind. With regard to sweets, in his early days he was under the spell of Miller, and did not believe in their harmful effect, but he now not only believed in the harmful effect of sweets, but saw their results in everyday practice. He believed the true etiology of decay would be found in the altered conditions of the foodstuffs. Mr. Hern had said that the chemical composition of foodstuffs had not altered, but that all depended on what was meant by chemical composition. With regard to the toothbrush, he naturally put it last because he did not think it was the first line of defence. With regard to gingivitis, that was not so much due to the fact that the one tooth was not rubbing on the other or feeble mastication as to the fact that the lips were not rubbing against the mucous membrane. He looked upon gingivitis of the six anterior teeth as absolutely diagnostic of mouth breathing, and it occurred whether there was protrusion of the teeth or not. With regard to rendering the mouth functional, probably the views of Mr. Hern and himself as to what was a functional mouth might differ. He himself believed a functional mouth was a mouth that would keep clean automatically without a toothbrush: it could only be functional if rendered in a condition to perform its function. The first thing, therefore, was to stop mouth breathing, and the second was to make the teeth fit to chew with. A small piece of root left in a child's mouth caused the child to bolt its food. With reference to Mr. Robbins and his sweets, he might suggest to him toffee; it contained a little dextrine and was far more prolific of dental caries. He knew of other distinguished practitioners who were in the habit of giving sweets to children, but he could not say that he agreed with it. With regard to Mr. Rushton's idea of dividing the paper into cases of rich and poor, surely in the treatment of disease there were the same principles underlying the treatment of the rich and the poor? Mr. Rushton had said that a hand-fed child was as well off as a breast-fed child, but he was sceptical on the point. He admitted that a child carefully brought up by hand might have good teeth, but a breast-fed child in some subtle way was a better child than a hand-fed one; it was not so prone, for instance, to get specific fevers, and it was well known that the death-rate from the exanthematous fevers in breast-fed children was nothing like so great as in hand-fed children. There was something in the milk of the mothers more than could be found by chemical analysis. At the present time he was engaged in an inquiry into the question of the methods of bottle feeding with regard to palatal deformities, and he asked that some of the members would follow that up, because there was very little doubt the hand-fed child did not in some way or other develop the same palate that was developed by the breast-fed child. With regard to Dr. Sim Wallace, he had made people think, and he himself thoroughly appreciated all the statements he had made that evening. He quite agreed, for instance, that a child could not masticate if it had not food requiring mastication. That was the whole secret: to give food that was hard and tough, and that required mastication. He had no time to enter into Mr. Stanley Mummery's point that it was a question of the survival of the fittest. It seemed to him to introduce the very vexed question of the transmission of acquired characteristics. If acquired characteristics were transmitted the ultimate results would be hopeless. To his mind the hopes for the race came from the fact that Weissman and others had shown that acquired characteristics were not transmitted, and if they were not transmitted then it was a question of some alteration in the environment. The environment in connection with the teeth was probably foodstuffs, and if the environment could be improved the race would develop teeth just as good as they were generations ago. To a man believing in the transmission of acquired characteristics the outlook on the dental question must be very pessimistic, but he was himself an optimist, because he believed that the nation was not degenerating, although it may have deteriorated, and he believed that the race might be as good as ever it had been provided it was placed in a good environment. Hope had brightened ever since the theory of the transmission of acquired characteristics had been thrown over. He felt that the question of the deterioration of the teeth was a most vital one, and unless the nation pulled itself together and endeavoured to find a remedy the position a half century hence would be very bad.

Mr. BENNETT'S resolution was then put and carried.

9.

Odontological Section.

April 27, 1908.

Mr. H. Lloyd Williams, Vice-President of the Section, in the Chair.

A Contribution to the Study of the Movements of the Mandible.

By Norman G. Bennett, M.B., L.D.S.

The movements of the mandible have been the subject of much investigation during the last few years, and several communications on the subject have been made to this and other societies. The contribution which I propose to place before you this evening is somewhat fragmentary, and consists mainly of an account of some experiments which I undertook some time ago, but have not had time to carry out to a full extent. As these experiments, however, were mainly directed to the elucidation of one or two points which did not appear to me to have been adequately dealt with by other workers, I thought they might be of interest to members of this Section and possibly stimulate further work on similar lines.

I do not propose to discuss in detail former communications on this subject, with most of which you are familiar; but I must briefly refer to the particular aspects of the problem upon which it did not appear to me sufficient light had been shed; and by avoiding the introduction of names I hope to escape from the need of subsequently supporting in the correspondence columns of the dental Press any opinions I may venture to offer, or criticisms of the statements of others which I may be compelled to make, in connection with this thorny problem.

In the first place it has been shown that the shape and curve of the surface with which the interarticular fibro-cartilage articulates varies considerably in different races and in different individuals of the same race; secondly, that the movement of the mandible does not consist simply of rotation about the condyle; thirdly, that the curves formed by successive positions of the moving condyle or, in brief, the path of the condyle, vary considerably in different individuals, as, indeed, would be expected from an examination of the dry bones.

So far as I know, no correlation between the movement of the condyle and the movement of the chin or symphysis, or indeed between any two points on the jaw, has been ascertained or exhibited. Furthermore, a fixed centre of rotation, outside the condyle, for the ordinary movements of opening and closing, has been supposed to exist, and much discussion—futile discussion, I may venture to call it—has taken place as to its exact position. I shall try to show you that no single fixed centre exists, but that the centre of rotation is constantly shifting. A path may be marked out showing the successive positions of the instantaneous centre of rotation for any given movement of opening or closing, and this path varies with the character of the movement.

In order to discover the exact correlation between simultaneous positions of the condyle and the symphysis in one individual, I made a somewhat elaborate apparatus for use in my own case. It so happens that, on account of the loss of lower molar teeth on each side, it was possible to construct a kind of rough metal plate like the bar of a lower denture, to fix firmly to the teeth by means of ordinary bands, and having wire extensions passing behind the premolars and round the buccal surfaces of the canines and incisors and meeting in the median line. By this means I found it possible to obtain quite firm fixation without in any way interfering with normal and complete occlusion. The wire extensions of the pseudo-denture were soldered at the point of meeting and passed outside the mouth with a suitable curve so as to interfere as little as possible with the movements of the lips. To this was attached an outside framework of wire with a side piece in a vertical plane parallel to the median plane and just outside the right condyle. Two very small incandescent glow-lamps were then attached, one immediately opposite the condyle and one opposite the sulcus below the lower lip. Here again I was favoured by Nature, because my condyles are thinly covered and can be exactly located with extreme ease, so that there was little difficulty in fixing the lamp with the tiny filament opposite the centre of the condyle. The exact position of the other lamp was of minor importance, because, as I shall presently show, a second lamp in any position is equally satisfactory; the lamps were connected with a small dry battery by means of fine, light insulated wires. The next step was to fix my head so that there should be no movement except that of the mandible. For this purpose I sat in a strong wooden chair having a high back and a kind of projecting wooden canopy with an oval hole cut in it to receive the crown of my head as far as the upper part of the forehead in front and the most prominent part of the occiput behind. I found, however, that to drive my head into this sufficiently hard to be firmly fixed was distinctly unpleasant, and it was necessary to interpose some adaptable material. An old and soft straw hat answered the purpose admirably; and I was then able to sit with all the apparatus fixed, and with my right side opposite to and parallel with a bare wall, in moderate comfort. Between myself and the wall was interposed a biconvex lens arranged on an adjustable stand. A large sheet of paper was pinned to the wall, the room was darkened, and the connection with the battery was made. The lens was brought into focus and the images of the two lamps were plainly seen, upside down of course, on the paper. The magnification depended upon the distance between the lamps and the wall. A convenient adjunct was an ordinary looking-glass placed in front of me at an angle of about 45 degrees, so that I could myself see the lamp images on the wall.

In this way I was enabled to open and shut my mouth slowly or rapidly and in a natural manner and to varying extents. While I did this my brother marked a series of corresponding points on the paper where the images of the two lamps for the moment rested, and we obtained the large diagrams I., II., III., which you see hung up. We afterwards obtained the diagram IV., to show lateral movements; for this purpose I, of course, sat facing the wall.

Now it is obvious that as the distance between the lamps is fixed, and is, in fact, 8·8 cm., or $3\frac{1}{2}$ in., the length between the pairs of corresponding points representing the two images should also be constant, and this fact afforded a useful check on the accuracy of the diagrams. We found that, although the magnification was considerable, the variations in this constant were very small; but the charts I., II., III., IV., prepared from the diagrams by means of the pantograph, have been slightly corrected by this constant; these have been photographed, and I shall presently show them to you on the screen.

I pass on to consider next the whole question of the centre of rotation. I am dealing now with movements of the mandible only in a vertical plane, that is to say, with positions which the mandible can assume without lateral movement. The mandible is, even in this limited respect, still capable of two independent movements, one being an angular rotation about the condyle, and the other being a translational movement (with no angular rotation), produced by the gliding of the condyle in its path. In the execution of any natural movement it is exceptional to find either of these simple movements alone; they are usually combined. Hence it results that the centre of rotation about which the mandible may be regarded as turning, at any instant, has a position not only different from that of the condyle itself, but capable of considerable variation with the nature of the movement.

As a preliminary to the attempt to follow the shifting centre of rotation during the normal opening movement of the mandible, it may clear the ground if I give a short discussion as to the purely geometrical and mechanical theory of the centre of rotation. I will ask your attention to a small mathematical demonstration.

Let a sheet of paper be supposed to slide about in any manner on a table. Let two points, A B (fig. 1), marked on the paper be used to specify its position. Join A B, and let A′ B′, A″ B″, &c., represent the successive positions of A B produced by the sliding of the paper. Join A A′, B B′, and from the middle points of these lines draw perpendiculars until they meet at the point O. Then it is obvious that O A = O A′ and O B = O B′ and, of course, A B = A′ B′. In other words the two triangles O A B and O A′ B′ are identical, and the movement from A B to A′ B′ may be accomplished by rotating the triangle O A B about the point O as centre. Next join A′ A″ and B′ B″, and in the same way obtain the point O′, and so on with all the succeeding positions. It is clear that the points O, O′, O″, &c., on the table form a series of centres of rotation about which A B moves.

Next from the point O draw O S′ = O O′, and inclined to it at an angle equal to the inclination of A B to A′ B′, that is to say, the angle through which A B moves about O. From S′ draw S′ S″ = O′ O″, and inclined to it at an angle equal to the inclination of A B to A″ B″, and so on to the last position.

We have now obtained two polygons. Now imagine the polygon O S′ S″ to be traced on the paper. Rotate O S′ about O until S′ coincides with O′ and A B with A′ B′; again rotate S′ S″ about O′ until S″ coincides with O″ and A B with A″ B″, and so on; that is to say, the rolling of the one polygon on the other exactly reproduces the changes of position of the paper. In the case we have considered, the various positions of A B are taken at comparatively wide intervals, so that the lines joining the series of centres of rotation form a polygon; but if we imagine them very close together, then it is clear that the polygon will become a continuous curve. We have now arrived at this important truth, namely, that any form of continuous plane motion may be represented, or reproduced by, the rolling of one curve upon another. These curves are called "centrodes," and the successive points of contact are called the "instantaneous centres." The forms of the curves, of course, vary with the character of the movement, but for any particular movement two centrodes, uniquely associated with that movement, may be derived, and the rolling of one centrode on the other will reproduce that movement.

In order to make this quite clear, I have prepared a simple working model belonging to the figure which we have just discussed. It consists actually of two planes, the one sliding on the other. On the moving plane are drawn the line A B and the centrode O S′ &c., forming parts of the edge; and the fixed plane is the diagram from which the lantern slide was prepared (fig. 1). If you place the moving piece in its lowest position, with the lines A B on each plane coincident, and then move it slowly round to the right so that the two centrodes are always in contact, you will see that when S′ becomes coincident with O′, A B coincides with A′ B′, and so on.

We may now examine the charts derived from actual movements of the mandible. Fig. 2 represents the paths of the condyle and symphysis in normal opening and closing, and the crosses outside the latter path represent extreme forward and backward positions associated with different degrees of opening. The cross lines are numbered and lettered to show the corresponding positions of the two moving points.

In fig. 3, from the same diagram, are shown the positions of the centres, by rotation about which each position of the mandible may be brought into its next consecutive position.

You see here the paths of the condyle and symphysis and also the fixed centrode. The line A B in fig. 1 is in this case, of course, the line joining the two lamps at the condyle and the symphysis.

I will now pass round this working model derived from the chart I have just shown, and you will again see that the rolling of one centrode on the other reproduces the movement of the condyle and symphysis from point to point in the series of momentary positions of rest.

In fig. 4, again derived from the same diagram, you see as before the paths of the condyle and symphysis, but the fixed centrode or path of the series of instantaneous centres is shown as a continuous curve.

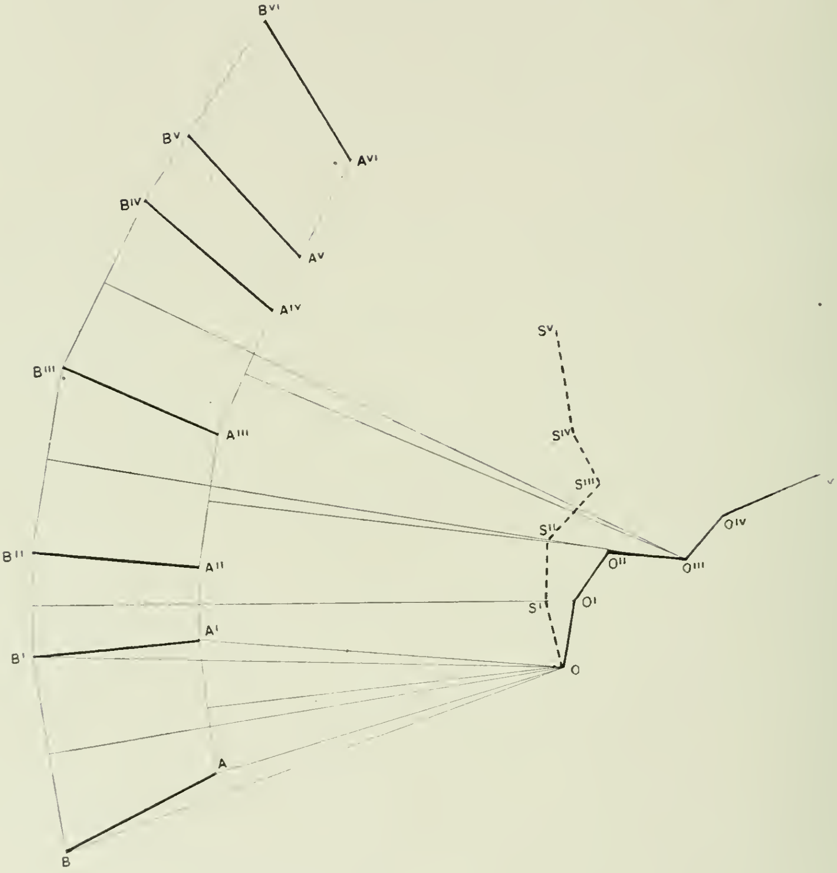


Fig. 1.

A B represent two points on a sheet of paper. A B, A′ B′, &c., represent successive positions of A B produced by the sliding of the sheet of paper on a table. O, O′, O″, &c., marked on the table, represent the series of centres of rotation about which A B turns. The movable polygon, or centrode, O S′ S″, &c., marked on the paper, if rolled on the fixed polygon, or centrode, O O′ O″, &c., will reproduce the several changes of position of A B.

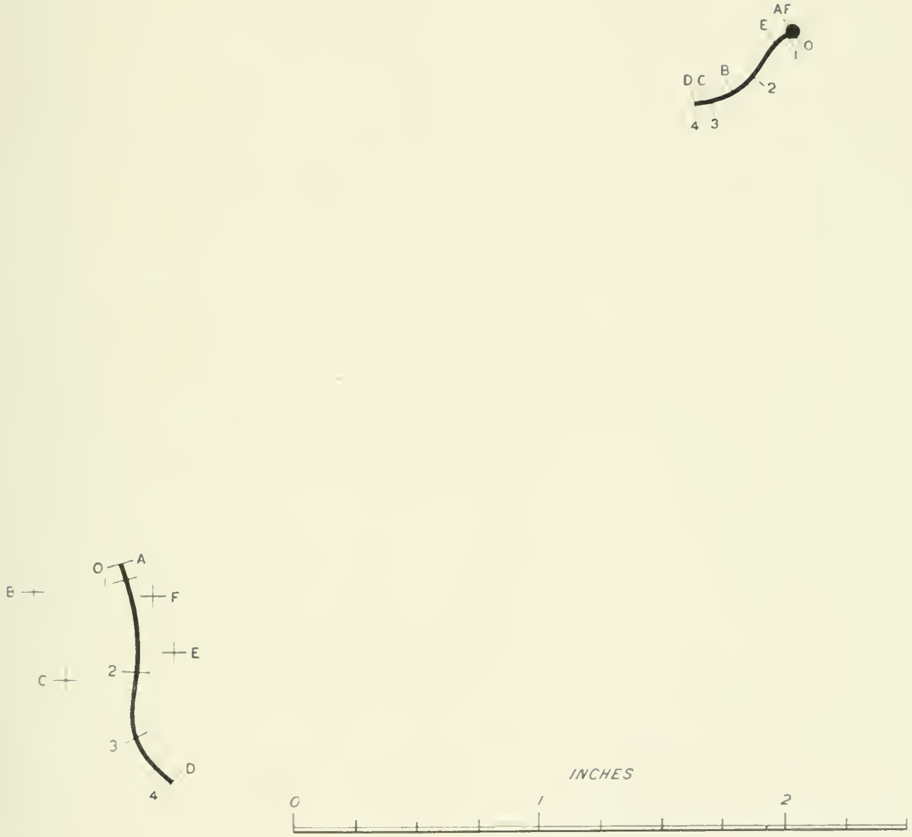


FIG. 2.

The upper curved line represents the path of the condyle and the lower curved line the path of the symphysis during the normal movement of opening and closing. The crosses represent extreme positions of the symphysis. The letters and figures indicate corresponding positions of the condyle and symphysis.

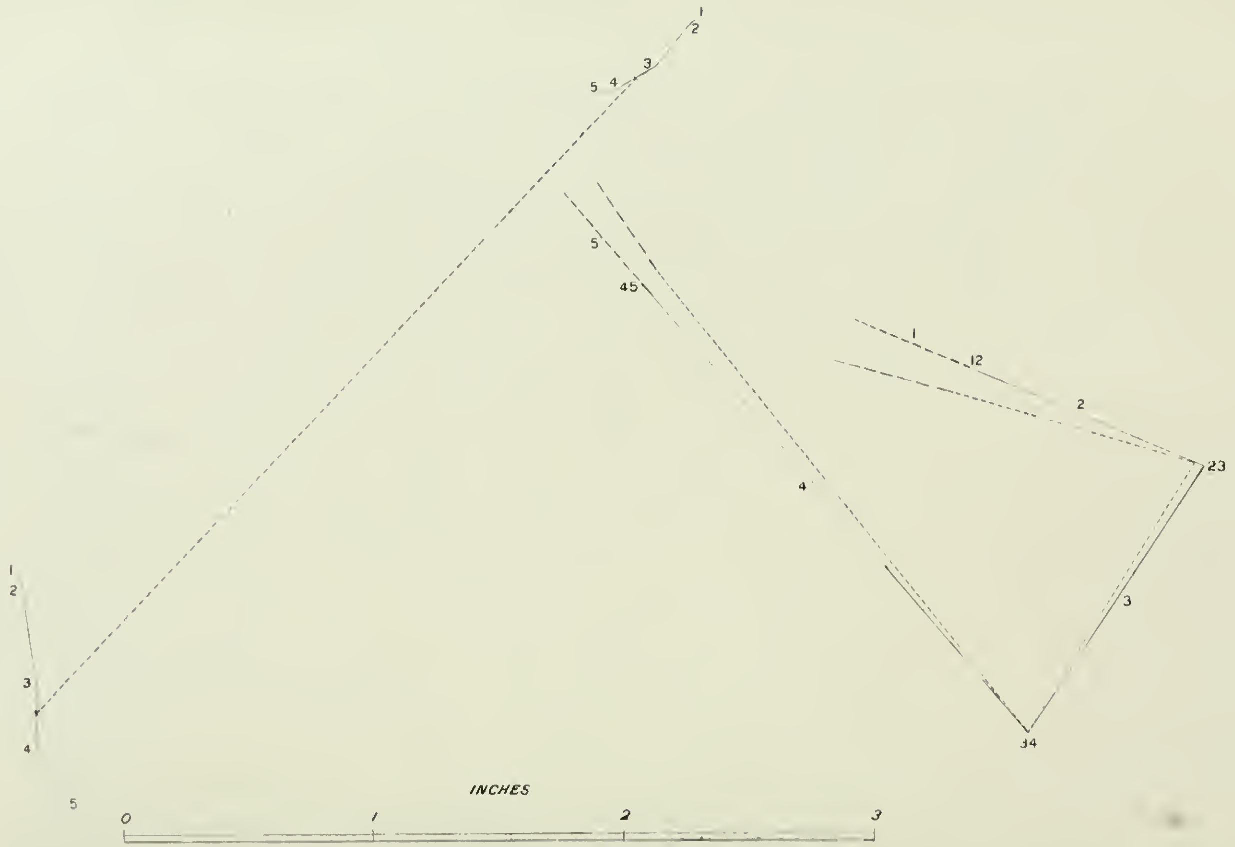


Fig. 3.

The paths of the condyle and symphysis are represented nearly as in fig. 2. The long, straight, interrupted line represents a line joining the condyle and symphysis. The two polygons shown by continuous and interrupted lines represent the fixed and movable centrodes respectively. The figure represents an intermediate position of opening, when the mandible is passing from the point 3 to the point 4, and the movable centrode is rotating about the point 34 on the fixed centrode.

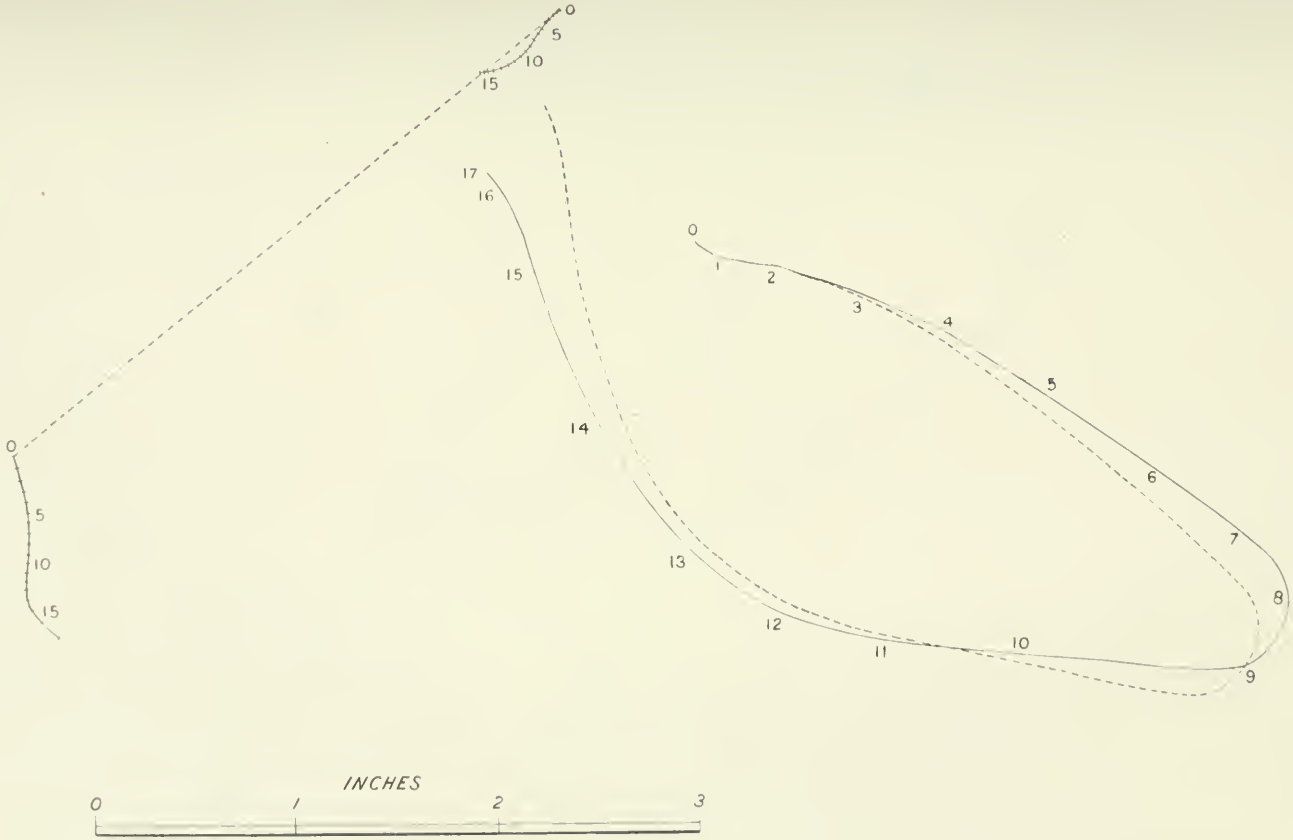


FIG. 4.

The paths of the condyle and symphysis are represented exactly as in fig. 2. The long, straight, interrupted line represents a line joining the condyle and symphysis. The two large curves shown by continuous and interrupted lines represent the fixed and movable centrodes respectively. The figure represents the position of complete occlusion of the mandible. If the movable centrode be imagined to roll on the fixed centrode, the two ends of the long interrupted line will pass through the successive correlative positions on the paths of the condyle and symphysis that correspond to the points of contact of the two centrodes.

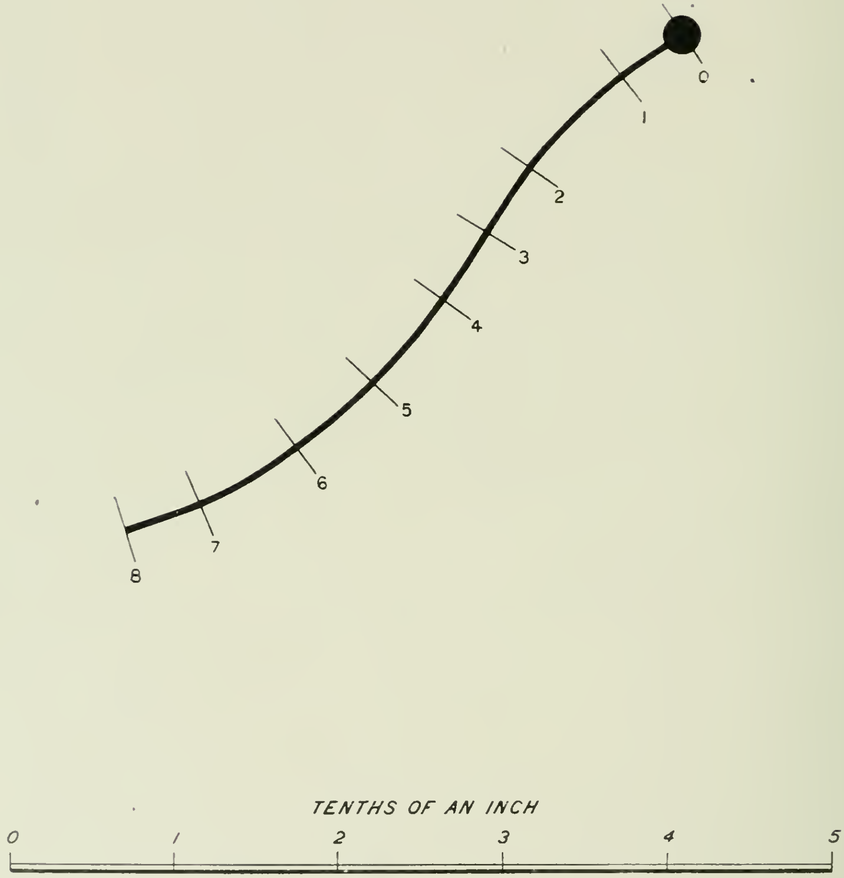


Fig. 5.

Path of condyle on a large scale.

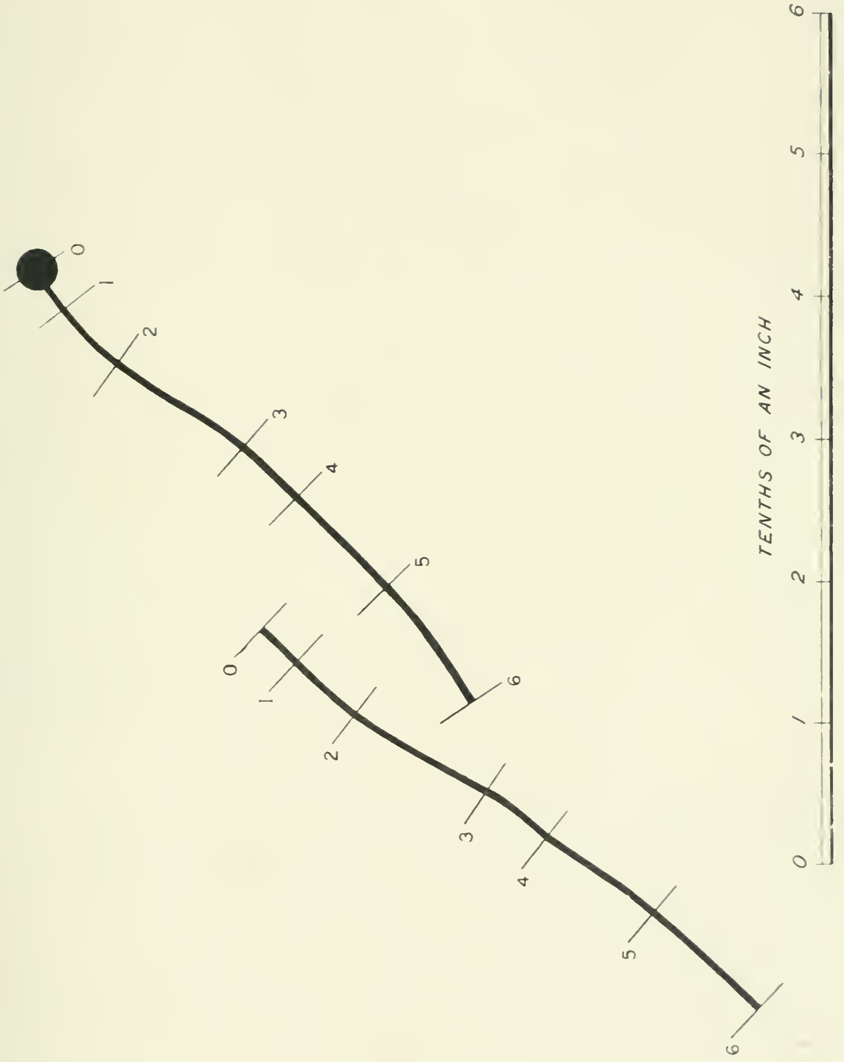


Fig. 6.

Path of condyle and of another point on the mandible in juxtaposition.

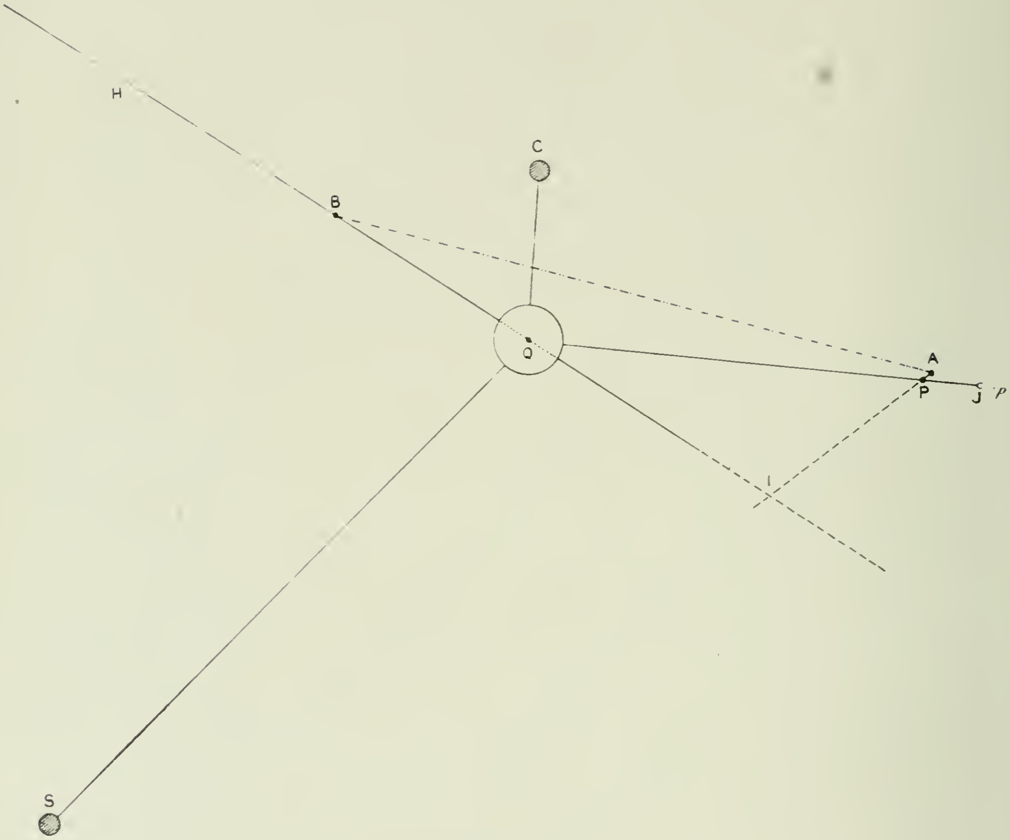


FIG. 7.

Diagrammatic representation of link-work mechanism. There are three moving pieces:—

(i.) The piece consisting of the three arms, Q S, Q C, Q P J, all rigidly connected at Q. This piece represents the moving mandible, C represents the condyle, S the symphysis.

(ii.) The link, H B Q I. It turns about the fixed point B and is linked to the mandible piece at Q. The end H is used as a handle to move the linkwork. (The end I is painted red.)

(iii.) The link, A P I. It turns about the fixed point A and is linked to the mandible piece at P. (The short link A P is almost concealed; its extension P I is the red pointer.)

The jaw, J, and the pin, p, ensure a smooth movement of the mechanism past the position in which the links lie along the line A B. The point, I, in which the lines of the two links intersect at any time, is *the instantaneous centre of rotation of the mandible piece.*

[P is moving perpendicular to P A, and therefore the centre of rotation for the mandible piece is somewhere on the line A P; similarly it is somewhere on B Q, hence it is at I.]

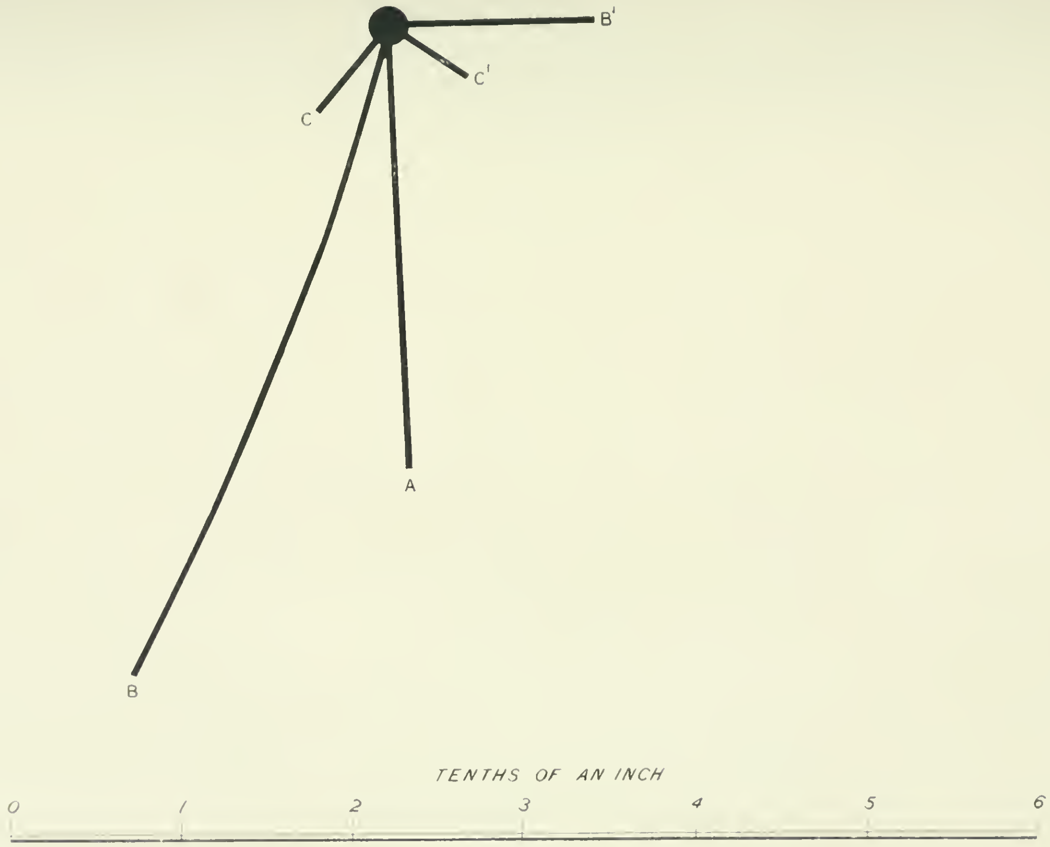


Fig. 8.

To show lateral movements of the condyle. A represents the movement of the condyle during extreme opening of the mouth without lateral movement; B during extreme opening, combined with extreme movement of the mandible to the left; B′ during extreme opening, combined with extreme movement to the right; C during extreme movement to the left, with the teeth in continuous occlusion; and C′ during extreme movement to the right, with the teeth in continuous occlusion.

This has been obtained by making use of the whole curved paths of the condyle and symphysis. In other words, whereas the polygon shows a series of a definite number of instantaneous centres corresponding to the series of momentary positions of rest of the mandible, and joined by straight lines, the curve shows an infinite number of correlative positions passed through by the condyle and symphysis.

I will now pass round another working model, and you will again see that the rolling of one centrode on the other perfectly reproduces the movements of the condyle and symphysis, or, in point of fact, the model moves exactly as the mandible did in my head and as its movement was shown on the wall. We see, then, quite plainly that there is no one centre of rotation for the mandibular movement, but that the centre is constantly shifting. It starts at a point behind and below the condyle, travels backwards and downwards, then forwards, and finally upwards and forwards, finishing at a point a little below the condyle path.

Fig. 5, from a different observation, shows the condyle path on a much larger scale.

In obtaining the diagram from which fig. 6 was derived, the second glow-lamp, instead of being fixed opposite the symphysis, was placed close to the condyle lamp, so as to give a larger magnification than in diagram I. From these two paths centrodes could be drawn which would perfectly reproduce the movement, and if the position of the symphysis were noted with the jaw closed, its movements would follow. In other words, for the purpose of reproducing the movement of the mandible, the paths of the condyle and of any other point are sufficient.

I have now to show you a piece of mechanism designed to exhibit, by means of a simple linkwork, the general characteristics of the movement of the mandible executed in opening and closing the mouth. It shows (fig. 7) the condyle, C, and the symphysis, S, moving on paths of some such shapes and lengths and relative situation as are found to occur in practice. It shows also for every position of the mandible the particular position of the instantaneous centre, I, about which the mandible is at the moment turning. The scale of the mechanism is a little more than 1 ft. to the inch.

Let us now return for a moment to fig. 2, and take note of the correlative positions shown. In the first place it is seen that even for very small openings there is a small forward movement of the condyle, that is to say, simple rotation about the condyle does not occur. In the second place, when the position of extreme opening is nearly reached, the condylar movement is small in comparison with that of the symphysis. In fact, the condylar movement appears to be somewhat the greatest, relatively to that of the symphysis, during the intermediate position of moderate opening.

Now the normal position of rest of the mandible is with the teeth slightly separated, but with the lips easily closed. When watching the formation of the curves on the wall during the experiments I took particular note of this position and tried it several times. The first cross mark on each path shows the position of rest, which is a very definite one as judged by subjective sensation; and it is after passing this point that the movement of the condyle seems to become rather suddenly considerable, although there is movement of translation from the very commencement, and the initial centre of rotation is, as I pointed out, below and behind the condyle. This fact seems to me to bear distinctly on the question of altering the height of bites in articulators. I mean that if a bite be taken which is found to be too much open and the height is reduced in a hinge articulator with the hinge correctly placed in relation to the plane of occlusion, errors will be introduced and the lower denture will necessarily be found to be placed too far back; but if the alteration be made in an articulator with the hinge nearly or quite in the plane of occlusion, then the reverse will happen. I mentioned just now that the crosses placed outside the curve, representing the opening and closing movement of the symphysis, indicate extreme forward and backward positions, and it is interesting to note the positions on the condyle path (marked by the same letters) that correspond. The oval curve which might be drawn through these positions of the symphysis show the movement of the mandible obtained by first pushing it forward as far as possible with the teeth in occlusion, then bringing it downwards, then backwards to the extreme limit, and finally upwards and a little forward to reach the position of normal occlusion. The oval curve therefore includes within its area all possible positions of the symphysis.

I will now place on the screen fig. 8. When obtaining this diagram I was sitting facing the wall in order to show any lateral movements of the right condyle. The line A, which is nearly vertical, is produced by extreme opening without lateral movement. You will notice that it deviates slightly to the right; this is somewhat interesting, because I am slightly larger throughout on the right side, and apparently the want of symmetry exists also in mandibular movement. The line B is produced by extreme opening combined with extreme movement to the left. The lines C, C′, belong to extreme lateral movement, with the teeth in continuous occlusion, to the left and to the right. C′ is interesting as showing that, in my case at least, in movement towards the side on which the condyle might be expected to remain stationary there is a quite considerable movement of the condyle outwards away from its articular surface, and slightly downwards. These two movements represent as nearly as possible those of normal mastication. The line B′ is that of extreme opening combined with extreme movement to the right. The condyle again leaves its articular surface to quite a considerable extent, the movement amounting to almost exactly 3 mm.

Now there is one very obvious criticism of these experiments, namely, that they are made upon one individual, and that therefore no general conclusions may be drawn from them. That is perfectly true, but I have carefully refrained from making any general statement as to the movement of condyles. These observations are, however, accurate within very narrow limits, and the work of other experimenters has shown pretty well to what extent the movements of the jaws and the paths of the condyle vary in different individuals; and, although these variations have been shown to be considerable within certain prescribed limits, the movements all have the same general character. Our experiments were conducted especially with reference to the supposedly fixed centre of rotation; there is no such point. If experiments similar to those I have described were made on another individual the paths of the condyle and symphysis would doubtless be found of somewhat different form, and the centrodes necessary to reproduce the movement would be also different; but that there could be for any individual a single centre of rotation is quite impossible, unless his condyle never left its position of occlusion, and then it would be in the condyle itself: or unless the paths of the condyle and symphysis were arcs of concentric circles, and this would involve very abnormal anatomical configuration.

I will now indulge in a few speculations as to the bearing of what I have said on the possibility of constructing an ideal articulator.

In the first place it would be necessary to obtain several correlative positions of both condyles and some other point in a simpler way than the one adopted, even though less accurate. It is conceivable that this might be done by means of a number of bites of different heights, each with an outside framework to register the several positions of the condyles. From these the centrodes could be obtained from which to reproduce the movements, and a working articulator might possibly be constructed to imitate Nature.

Secondly, the models of the jaws must be attached to the articulator in such a way that they bear the same positions relatively to the line passing through the two condyles as the jaws themselves. This may be done with the face-bow in connection with any one of the bites taken. In this way it might be possible to reproduce the natural movement of any individual mandible, but there would yet remain the question of finding the correct height of the bite and the correct plane of occlusion. These two considerations are really distinct from the question of the construction of an ideal articulator, and are connected with the fact that in edentulous and many other cases disease has destroyed the normal conditions.

After all, I do not feel at all convinced that the ideal natural articulator is a *sine qua non* for practical prosthetic dentistry. In constructing artificial dentures we are concerned only with the smallest degrees of openings and with lateral movements with the teeth in occlusion, and I believe that for these purposes a small movement of rotation and translation, combined with some lateral freedom of the correct kind, would probably be sufficient, if there were a scientific method of fixing the plane of occlusion in relation to the condyles when the normal occlusion has been lost. On this point I hope to have something to say at a later time, but, as several years have elapsed since I made the experiments which I have only now described, I am able to assure you that you need have no fear of being troubled before a somewhat remote date.

In conclusion, I should like to say what you have already probably suspected, namely, that this paper would never have been written by myself unaided. I am greatly indebted for his assistance to my brother, Mr. G. T. Bennett, Fellow and Mathematical Lecturer of Emmanuel College, Cambridge. He collaborated with me in the experiments themselves, instructed me in the mathematics bearing on the problem, and drew the charts derived from our direct observations; and although I have managed to write the paper itself, he has also kindly revised it and saved me from making any confusion in mathematical expression. The problem is, in fact, more geometrical than anatomical, and I cannot help thinking that more frequent collaboration between workers in different fields, meeting on border-line subjects, would result in the elucidation of questions which present much difficulty to the specialist. It is one of the inherent weaknesses of specialism that its exponents must become limited to following the natural trend of their own habits of thought; and it is indeed very refreshing to find that just that aspect of a problem which presents most difficulty to the solitary worker is really only a special case of a general truth well known to a student of another branch of science.

DISCUSSION.

Mr. F. J. BENNETT said all the members appreciated the carefully thought out paper, and if full and ample criticism was not offered it was really because they preferred to wait and read the paper when printed. He endorsed what the author had said about the advisability of collaborating with men engaged in other walks of life, because he felt sure that a good many things which were now mysterious would then be made plain. It was perhaps a little disappointing to find the author hinting that, after all his elaborate inquiries, perhaps the empirical method of obtaining a bite was the safest course to follow. That, he presumed, was intended merely for the moment, because no doubt a more rational method would be gradually devised, and he was confident it would largely be dependent on such work as the author had brought forward. He thought that in thanking Mr. Bennett for his valuable communication, thanks should also be accorded to his brother for the assistance he had afforded in preparing the paper.

Mr. E. LLOYD-WILLIAMS declared that his enjoyment of the paper had not been lessened by the fact that he had been unable to follow a great number of the mathematical descriptions. He was wondering how many others were in that same position; but, although he could not follow the mathematics, he was somewhat relieved at the end of the paper to find that, after all, in the practical application of the subject, the obtaining of an accurate bite in an edentulous case was not dependent on any anatomical articulator. He had always suspected it himself in spite of the very many learned papers that had been written, and had felt satisfied that the practical man who could produce the normal occlusion at rest was the man who was going to succeed in making a full set of teeth likely to be useful to a patient. The important matter was the normal point of rest in occlusion in every edentulous case, with a certain allowance for lateral motion, and those things, he thought, were not to be obtained so much by the help of an articulator as by the practical application of one's experience and knowledge when working in the mouth. He was interested to find that Mr. Bennett was of opinion that no amount of calculation would ever show in an edentulous person well past middle life what the normal plane of occlusion was in adult life when the patient possessed all the natural teeth. That must be left to the dentist's imagination. It was there where his art might rise a little above the common level. The success in adjusting a set of teeth in an edentulous mouth must in nearly every case depend very much upon the personal equation, the art and imagination of the dentist treating the case. He hoped Mr. Bennett would follow the matter up and bring before the Section in the near future the practical results of the very careful theoretical work which they had been privileged to listen to that evening.

Mr. RUSHTON thought it would be an extremely interesting matter if the author could carry out the same series of experiments in some edentulous cases. Those were the cases that had to be dealt with in every-day life, and they often differed considerably from the normal. If a certain range could be found, people could be also found of sufficient mechanical ability to make an articulator with the required movements. A great advantage would be gained, and he did not see any great difficulty in the movements being reproduced mechanically.

Mr. GABELL congratulated the author on having placed before the Section a paper which, although mathematically very complex, was very illuminating as to the actual movements of the jaw. The models and diagrams afforded a most wonderful exposition of a very complex mathematical subject brought down to plain matter of fact which everybody could understand. He should like to have some information with regard to the use of the lens in obtaining the diagrams, as it seemed to him to require a rather huge lens. He also wished to know whether it was practicable for other people also to produce diagrams without expensive apparatus. He agreed with Mr. Lloyd-Williams that there was not yet an articulator which helped very much in setting up teeth, but he hoped in time something would be obtained of assistance in discovering the movements of the jaw and in arranging the curves of occlusion. Mathematics, he thought, would prove of great aid in that direction.

Mr. H. LLOYD WILLIAMS had been very much interested in noting the path of the jaw when it was pushed back as far as possible so as actually to enable one to obtain a tracing behind the normal. Practically he had adopted the view that one could not bite behind the normal to a sufficient extent to upset in any way the articulation ; but some cases he had met made him doubt that, nevertheless, as a principle of general application in the taking of the bite it remained quite reliable.

Mr. DOWSETT thought the author had once and for all settled the question that there was no fixed centre of rotation. He was sorry to hear a man so eminent as Mr. Lloyd-Williams sound such a pessimistic note with regard to an anatomical articulator and rather decry the necessity for it. Although the position in which dentures were required to occlude was nearly the position of rest of the condyle, nevertheless when the dentures were being used it was the lateral motion that had most to be considered, and it was just that lateral motion, although very slight, which caused a denture, otherwise beautifully constructed, to tilt. To overcome that tilt it was absolutely necessary if possible to be able to reproduce the lateral and forward motion in an anatomical articulator, not merely relying upon the dentist's skill and experience. He saw no reason why such an articulator should not be eventually constructed.

Dr. SIM WALLACE said the tracings had been taken in connection with a bite where there were teeth, but he imagined it was possible that the jaw might rotate from a fixed point after the normal occlusion was passed in the case of an edentulous person. Consequently it was possible to imagine that if a close bite were taken there would be some justification in using a hinge articulator to open it.

Mr. NORMAN G. BENNETT, in reply, dealt first with the connection of scientific investigation with practical problems. He thought it was not always possible, immediately after ascertaining some definite facts with regard to definite anatomical movements, to] say at once that that knowledge enabled such and such things to be done; but, on the other hand, he did not think anybody could say that it would never enable some practical improvement in methods to be brought about. Dentists were more or less anxious to have an anatomical articulator, and any information with regard to the movement of the jaw must be of some use in obtaining such an instrument. It was on those lines that many practical improvements had been made, made often at a date very remote from the scientific discovery. Most practical improvements in machinery and other things had been founded on scientific investigation, and that fact was a justification for trying to work out a problem of the kind under discussion. He thought it was quite natural that many members were unable to follow the mathematics at the moment; this would be much easier in print than in oral description. The object aimed at was, firstly, to get actual tracings from life of the two paths in correlation, and not merely of some particular point, and secondly, to reproduce the movement in the models, and thirdly, to show that the centre of rotation, which was supposed to exist somewhere below and behind the condyle, did not exist as a single point but as a curved path. With regard to articulators, there were two points which were very often confused. An ideal articulator was one which would reproduce the movements of the jaw of any particular individual, and he did not think there would be any great difficulty in constructing an ideal reproduction for the one particular patient. But the ideal articulator required was some piece of mechanism which might be very readily adapted from one person to another, an apparatus reproducing movements in different individuals. That seemed a fairly large order, and he thought it would be a long time before it was done, although he thought it might be done in time. That, however, was quite distinct from the question of placing the models on the articulator in the same relative position to the articulator, or to the line joining the condyles, as obtained in the mouth. That having been done, the height of the bite had to be fixed, and he believed the question of the height of the bite was capable of having very much more light thrown upon it than it had at present. At present, as Mr. Lloyd-Williams said, the height had to be gauged more or less by imagination. It was on that particular point that he hoped to say something at a later date. With regard to apparatus, it was not necessary to have a large lens. On first writing the paper he thought a fairly large biconvex lens was a point of some importance, but his brother had shown him that a small lens would do quite as well. He thought the point raised by Dr. Sim Wallace was very likely true. In an edentulous patient there were two new conditions: no teeth and slackened ligaments around the joint. A patient might be able to close his mouth further than the original position of normal occlusion, and if in normal occlusion the condyle was as far back as it would go, it followed that the movement beyond that would be a movement of pure rotation around the condyle, unless the slackening of the ligaments allowed the condyle to move backwards, when there would also be a translational movement. The movement of the condyle in old people was freer than in young people. He hoped at some future time to deal with the question from the more practical dental aspect.

A Case showing Incomplete Eruption of the Temporary Molars.

By H. W. TREWBY, M.R.C.S., L.D.S.

THE case was illustrated by a large number of photographic slides. The patient first came under Mr. Trewby's care at the age of 8. Although of delicate constitution she had never been ill, and there was no history of rickets or adenoids. Her condition showed that she had a symmetrical separation to the extent of more than $\frac{1}{4}$ in. on both sides of the jaws between her temporary molars (figs. 1 and 2). The first

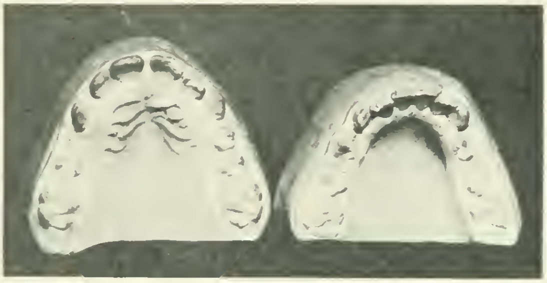


FIG. 1.

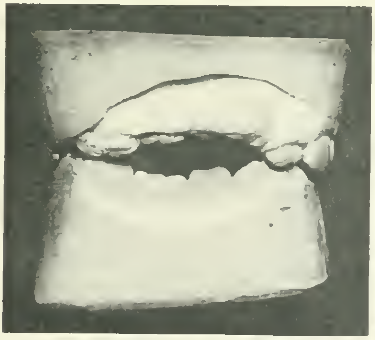


FIG. 2.

lower temporary molars were lost and the first bicuspids erupted, but an X-ray photograph showed that none of the other permanent bicuspids were in the jaws. At the age of 12, as the teeth were then nearly buried in the gums and caused considerable inconvenience to the patient, all the four uppers and the two lowers were extracted and found to be entirely absorbed, being represented only by the crowns. Mr. Trewby mentioned that Mr. Colyer had had a case of a somewhat similar character, of which he showed illustrations.

Mr. J. F. COLYER said it would be interesting if other members could say whether they had met with similar cases. Such cases, although uncommon, were, he thought, often missed in practice. The point was to try and find out what was the actual cause of the want of growth in the deciduous molar region. In the case he had lent to Mr. Trewby the interesting point was that, in addition to a deficient eruption of the deciduous molars, there was also a stunted eruption of the right mandibular first permanent molar.

The HON. CURATOR (Mr. J. F. Colyer) exhibited a series of slides made from photographs of specimens recently added to the Museum.

Odontological Section.

May 25, 1908.

Mr. J. Howard Mummery, President of the Section, in the Chair.

Notes on the Extraction of an Upper Wisdom Tooth.

By Wm. Rushton, L.D.S.

Most of us have had unpleasant experiences with extractions of lower wisdom teeth, and the reasons to account for these are obvious. The tooth is often firmly impacted between the ascending ramus of the mandible and the second molar, and the bone is dense and unyielding; therefore great force has often to be employed. Furthermore, the action of gravity does not assist Nature in getting rid of pus or septic matter. These factors are absent in the case of the upper wisdom tooth, and consequently the latter does not bear the evil reputation of the lower wisdom tooth. That great pain and constitutional disturbance can, however, occur in the case of extraction of the upper third molar tooth will be seen by the case I shall bring before your notice. I bring the case before you for several reasons. One is that I do not know the cause of the pain and inflammation occurring in connection with this perfectly sound tooth, nor the cause of the prolonged suffering which followed extraction. I do not know whether the same chain of symptoms would have supervened had the tooth not been extracted, or whether—in the light of the suffering endured by the patient—extraction was the best course of procedure. Further, I should like to gain any knowledge which would enable me or others to obviate or reduce such pain in any similar case in future.

Lastly I wish to place the case on record from a forensic point of view. Fortunately my patient was a man of refinement and intelligence, who gave me credit for doing my best for him under the circumstances. But all patients are not cast in that mould, and the increasing number of actions at law against members of our profession makes it incumbent upon us to record and discuss such cases so that we may be able to answer any charges brought against us in the exercise of our profession.

On March 25 I saw the patient, a healthy, vigorous man aged 53. He had had pain and swelling round the left upper wisdom tooth for several days, and, on the morning on which I saw him, had great difficulty in opening his mouth. The general condition of the mouth was clean and healthy, but he had had slight pyorrhœa for some years affecting the lower incisors. This had been successfully kept in check and there had been no recent discharge. The left upper second bicuspid had been crowned many years previously and the root was gradually becoming detached from the alveolus. The wisdom tooth had never properly erupted, the surface of the crown being level with the gum. As the tooth was functionless I advised extraction, which was accomplished after I had injected $\frac{1}{3}$ gr. of cocaine hydrochlorate in ♏ xxx. of water. No undue force was exercised in the extraction; in fact both patient and myself were surprised at the comparative ease with which the tooth was removed, especially considering that it was not properly erupted and that the root was large and bulbous.

The sequel to the extraction was increase in the pain and trismus from which the patient had been suffering, in spite of his using infusion of poppy-heads. Sleep was only obtained by means of sleeping-draughts.

On March 30 patient remained at home, exhausted with nights and days of pain. On March 31, having some important work, he returned to his office, but severe pain continued until April 6, when I again saw him. It was difficult to examine the mouth as the patient could scarcely open it, but I could see no discharge. I prescribed a local anodyne and antiseptic mouth-wash.

On April 7, the pain and trismus being no better, I took him to consult Mr. Pearce Gould, who made a digital examination. Mr. Gould said that the pain proceeded from the tuberosity, but could discover no fracture, and prescribed rest in bed, aspirin, and mouth-washes of hydrogen peroxide and Sanitas. By this time there was cellulitis as far as the neck and much difficulty in swallowing. The digital examination much increased his pain and he could only obtain snatches of sleep by means of sleeping-draughts.

On April 12 the pain began to diminish, and on April 21 he had the crowned bicuspid extracted by Mr. Dewes, which seemed to give further relief. The next day he went into the country for rest and change. He still had great difficulty in using his jaw for eating, but this gradually improved. He had had no increase of temperature throughout. He had lost 12 lb. in weight, probably partly due to inability to masticate.

This, gentlemen, is a brief description of the sequelæ of what appeared to be a simple operation, but which proved to be, in the patient's own words, "the longest and most painful illness he had ever experienced." If I might venture to theorize I would conjecture that some septic organisms had invaded the peridental membrane, perhaps induced by the abnormal position of the tooth, and that the extraction, in spite of aseptic precautions, had possibly augmented the invasion. I do not attribute any importance to the injection as a factor. The injection was performed well outside the peridental membrane and there was no sloughing. It is also probable that, in the condition of trismus prevailing, the stretching of the muscles in extraction and subsequent examination increased the pain.

DISCUSSION.

Mr. J. G. TURNER asked why the needle of the syringe should not be held responsible. It probably passed through an infected area and carried germs deeper still, and the whole thing was an infective septic osteitis and periostitis.

Mr. KENNETH GOADBY did not agree with Mr. Turner, because it was an unusual thing to get any marked degree of cellulitis in the mouth, with septic organisms present, without a rise of temperature. He did not know of any case of an osteitis of that kind due to streptococci, staphylococci, or pneumococci, without a distinct rise of temperature. He was inclined to regard the case as one of toxic origin.

Some Clinical Notes on Pyorrhœa alveolaris.

By J. G. Turner, F.R.C.S.

There seems to be some danger that clinical observation may be neglected in our newly awakened belief in the omnipresent bacterium, and, somewhat in the hope that clinical observers will not relax their efforts, I bring forward these notes.

Etiology.—Gout has long held the palm as a cause of pyorrhœa, many seeming to regard pyorrhœa as a sign of gout. If gout be pyorrhœa and pyorrhœa be gout, then this undoubtedly is so, but the cases I see present no other signs of gout, and I doubt if those who so facilely tell their patients they are suffering from "gouty gums" ever see any other. I can recall but two cases in which affections which might have been considered symptomatic of gout coincided with pyorrhœa. In both these the big toe was attacked, and both got well only after extraction of every tooth; so with every other diathesis credited with producing pyorrhœa. Myself, I believe it to be a local disease, influenced secondarily by other causes of bodily depression. The incidence of pyorrhœa among the teeth has an important bearing on its etiology. I believe it is generally accepted that it begins among the front teeth (and I think Mr. Goadby adopts this view); but I have found that in a very large number of cases, perhaps 50 per cent., it is most advanced among the molars, *i.e.*, it probably began there. These are the teeth with the largest approximal surface and least accessible for cleansing. In several cases I have noted that the labial gum margins of the incisors, canines, and premolars have been free from pyorrhœa, which has obtained a serious hold on their lingual aspects and among the molars, and in each case the patient has been an assiduous user of the toothbrush. Obviously the parts most easily cleaned have escaped. These observations compare exactly with the incidence of dental caries on the more remote and less easily cleaned parts of the teeth, and I believe both to be essentially of the same character—dirt diseases. The "dirt" or pyorrhœa is made up of germs, with or without food debris, and stagnation is its complement.

Age.—Pyorrhœa is a disease of early adult life, seldom found in full development in young persons. The earliest age in which I have found it was at 14. Mr. Goadby suggests that pyorrhœa is due to milk infection: the age incidence, when but little raw milk is consumed, and the frequency with which it commences among the molars seem to render it unlikely that this is a frequent mode of commencement.

Infectivity of Pyorrhœa.—Clinical observation leads me to believe it to be in general of low infectivity. Such fleeting possibilities as are offered by pipes, spoons, kissing, &c., may be ignored, but there seems to be an acute form closely approaching ulcerative stomatitis of children in its clinical aspects, even to acute infection of the corresponding parts of the cheeks. I have seen this grafted on a pyorrhœa perstans during the dusty season and have regarded it as the result of a secondary infection. Bacteriologists may be able, some day, to tell us its true relation to ulcerative stomatitis, and some may have seen this form arising *de novo*.

Fragility of the Teeth.—I have long been struck by the ease with which pyorrhœa teeth, however loose, are broken in extraction, a portion of a root being very commonly left in. This does not arise from absorption, as I have verified by taking out the fractured piece. If we examine a large number of pyorrhœa teeth after extraction, teeth which have had a very considerable bony attachment left, we shall find apical absorption very frequent. This means that infection has progressed through the whole length of the periodontal membrane, and it may be that the nutrition of the dentine is altered by absorption of toxins via the cementum. The accompanying specimen, extracted for pyorrhœa, seems to show that the whole of the tooth may be affected; it broke with surprising ease on my closing the forceps, though I was forewarned by my belief in the fragility of such teeth.

DISCUSSION.

Mr. NORMAN BENNETT said the idea that gout was a cause of pyorrhœa was largely due to the fact that pyorrhœa was confused with premature senile absorption of the alveolus. He had a case quite recently of a patient who had been suffering from pain in both jaws for a considerable time, and he found that several of her teeth were loose. There was no pyorrhœa, and it did not appear that there had ever been any. The alveolus was largely absorbed—so much so that in an upper molar he could feel what he thought was the end of the palatine root, which was completely exposed for its whole length. The patient had had gout in the foot and regarded the case as "gout in the jaws." He removed two of the teeth and found the upper molar in a very peculiar condition: the inner surface of the palatine root and the ends of the buccal roots were absorbed, and had an appearance similar to a temporary tooth.

Mr. KENNETH GOADBY was not quite certain whether he was accused of saying that all pyorrhœas commenced in the front teeth, but he might have stated that it was a common site of the commencement of pyorrhœa. It did not follow, however, it was the only site. There were many points in a mouth where food might lodge, and those were the points where pyorrhœa might be expected to commence. He would not touch upon the question of local disease, because it was probably a question of local infection, and it did not matter very much whether it was called accumulation of food or direct local infection; but with regard to the milk question he had thrown out a hint that the bacteriology of ulcerative stomatitis in children and pyorrhœa alveolaris were remarkably similar. The incidence of milk-borne disease was an exceedingly difficult thing to explain. Infected milk might convey typhoid fever, scarlet fever, or diphtheria to a large number of children, but a still larger number of those exposed escaped the infection. Pyorrhœa might commence in early life as quite a chronic condition, and it was interesting that its first manifestation, according to Mr. Turner, should be always in early adult life, when one would expect rather that it would be likely to begin to make itself manifest. There was another point that rather led him to suppose that milk might be an associated factor, in that certain bacteria found in pyorrhœa alveolaris were undoubtedly bacteria that were borne by milk. About four years ago Klein showed certain specimens of yeast obtained from London milk. The milk had been inoculated into guinea-pigs and two of them had died of a species of disease which was found to be due to saccharomyces. Other observers had shown that pathogenic yeasts were not uncommon in milk, and he himself had succeeded in isolating in some twenty cases a highly pathogenic yeast from certain varieties of pyorrhœa. Pyorrhœas were not all the same, and one found that certain organisms were present in certain cases more than in others. The pathogenic yeast from the twenty cases he had identified with the yeast described by Klein, and it was one which bore considerable relation to the *Saccharomyces neoformans.* It was an interesting organism, because when inoculated into animals it produced a species of new growth in the kidney, lung and liver which on dissection was exceedingly like a sarcoma. When it was remembered that many pyorrhœa cases showed a considerable amount of local hypertrophied tissue before the alveolus commenced to atrophy, it was somewhat interesting to remember that the pathogenic yeast that might be obtained from the cases when inoculated into animals would definitely produce disease. It was a well-known fact that certain organisms closely related to the diphtheria bacillus were to be found in milk, and except by infected individuals, who were infecting the milk, there was no knowing where the organisms came from: they were not found in the soil nor were its allies. They were found, however, in butter or in cheese, and in both cases, of course, came from the milk. There were several diphtheria-like bacilli to be found in milk, and he had succeeded recently in isolating a diphtheria bacillus from several pyorrhœa cases which morphologically was similar to the diphtheroid bacillus and which was identical with one of the bacilli found commonly in infected milk. The ordinary

London milk in its unboiled condition contained somewhere about 5,000,000 organisms per cubic centimetre; and that was ordinary clean milk such as was distributed by the milkmen. Certain organisms could live under the conditions in the mouth and certain others not, so that although bacteria were present in the milk they were not found in the mouth in all cases. Miller washed his mouth out with a strong solution of lactic acid bacilli and made cultures at the time and twenty-four hours later, and in twelve hours time the lactic acid bacillus had entirely disappeared. With regard to milk being the carrier of pyorrhœa, he had not so definitely stated, but he saw no other channel by which the disease could be spread except milk and dust. A class of organisms that definitely contaminated milk was ordinarily found in sewage—lactose fermenters—and quite commonly found in pyorrhœa. They might have come from dust, but they were not found present in the air. With regard to acute cases, Mr. Turner was inclined to regard them more often as occurring as sequelæ or as a complication of chronic pyorrhœa. Mr. Goadby exhibited the temperature chart of a patient suffering from pyorrhœa and no other ascertainable disease. When he saw the patient first the temperature was 102° F., and she was sent to bed. She had nothing but red gums and sore mouth. Then came a tremendous drop in the temperature following the administration of 15 gr. of aspirin. It was found to be too depressing and its use was discontinued. She was found to have in the blood a certain number of streptococci identical with those obtained from the mouth, but the opsonic index with regard to the organism was normal. The temperature reached 105° F., and then he found a number of staphylococci in the mouth. Staphylococci were not so common in the mouth as some people supposed; he had found himself, as a rule, about 15 per cent., and Miller had found 18 per cent. On inoculating the patient with staphylococcic vaccine her temperature came down. Within forty-eight hours she had acute tenderness round the whole of the mouth, and in one spot a huge periosteal abscess, which was opened. She had a further series of staphylococcic injections, running up in the last case to 10,000,000,000,000, and finally the temperature came down and the patient was now quite well. During the two weeks her condition was exceedingly grave and was undoubtedly an acute uncomplicated case of pyorrhœa.

Mr. J. G. TURNER said that what Mr. Norman Bennett's patient thought was gout might be a dry caries of the alveolus, a very common form of pyorrhœa. With regard to Mr. Goadby, the question of where the bacillus came from or where it was found would not alter the fact of distribution in the mouth, nor would a laboratory argument be held proven unless it fell into line with the clinical. He still maintained that the fact that the front teeth, which were cut early and were in situ at a time when a fair amount of unboiled milk was taken, were not attacked in 50 per cent. of the cases, while the molars, coming after the milk was changed for some other diet, were commonly attacked, was against milk as the particular source, although the bacteria might be found in the milk.

Heredity and Dental Disease.

By Stanley P. Mummery, M.R.C.S., L.D.S.

Few branches of science have been more neglected by medical men than heredity. Of all the workers on this important subject nearly all have stood outside the ranks of our profession: Lamarck, Darwin, Wallace, Spencer, Romanes, Haeckel, and Weismann—to none of these great names can the medical profession lay claim. Indeed, the loose statements which one so often hears from the lips of medical men is a sign of the almost complete indifference towards this great question which is displayed by the profession at large. More especially does this observation extend to dentists, and a paper read by Mr. Norman Bennett before the Odontological Society in 1901, and more recently one by Mr. J. F. Colyer before the Students' Society, are the only contributions to this subject which I can find in dental literature for many years back.

The cause of this neglect is, I think, to be found in the commonly accepted idea that heredity plays little or no part in the causation of dental caries, and that no results of practical value could therefore accrue from its study. Never, I believe, has a more erroneous idea gained credence, nor one more likely to retard the progress of useful inquiry. There is no question connected with the human species upon which the facts of heredity will not throw some light. Dental disease, as I shall hope to prove to you to-night, depends for its very origin as well as increasing prevalence upon hereditary factors.

It is frequently stated that the true cause of dental caries has yet to be discovered, and that although we now know the pathology of the disease through the investigations of Dr. Miller, the etiology is a very different matter. The truth of this is obvious, and yet, with the notable exception of Dr. Sim Wallace, whose work I shall refer to again later, nearly all the searchers after the etiology have pursued further pathological studies in their endeavours to discover it. The teeth and oral secretions have been analysed again and again, even the urine and faeces have been examined to see if the cause lurks there. Everything, from milled bread up to sulpho-cyanide of potassium, has been blamed in turn; but I am convinced that the cause will not be discovered under the high powers of the microscope nor in the analyst's test tube. It is only in the study of Nature's laws, in heredity and natural selection, that the truth will be found.

Leaving, then, for the present all microscopical and analytical research, we must turn to the study of Nature herself, in the better understanding of whose workings lies the secret of many hitherto unsolved problems. I must ask you, therefore, to forgive me for leaving for a short time the immediate subject of my paper and dealing very shortly with the relations between heredity and disease in general.

In 1813 Dr. W. C. Wells read a communication before the Royal Society upon the differentiation which exists between certain races of mankind. This differentiation Dr. Wells explained from the fact that since no two individuals are alike, some would be better fitted than others to resist the diseases proper to a particular country, and would consequently tend to survive, whilst their less fortunate neighbours would perish in greater numbers. This communication was read years before Darwin conceived his theory of natural selection, and has therefore a double interest.

Quite recently Dr. Archdall Reid has taken up this theory of Dr. Wells, and in his book, "The Principles of Heredity," he traces the operation of the law of natural selection among civilized races, acting through the agency of disease. I wish to acknowledge my indebtedness to Dr. Reid, since his writings largely suggested to me the ideas contained in this paper.

A belief that is not uncommonly held is that natural selection has almost entirely ceased to exert any influence on civilized man, owing to his artificial mode of life. Of course, among us, the surest foot, the keenest eye, or even the most powerful brain, do not give the individual that superiority over his fellows that would enable him to leave a greater number of offspring, as is the case with wild man and animals. Nevertheless, one cannot doubt that Nature still exercises a great influence over her more educated children—civilized men—and even by the same old methods by which she caused their evolution from the lower animals, viz., elimination of the unfit. She employs the same old methods, but not quite in the same way, since the characters she once chose to select us by are now comparatively of little value to us in the struggle for existence; hence Nature cannot control us by dealing with these. In disease, however, she has a very efficient agent by which to mould us, and there can be very little doubt but that certain races have gained that natural immunity against special diseases, which we know them to possess, through the agency of natural selection. As examples I may mention the West African negro's immunity to malaria and the European's comparative immunity (as compared with the negro) to tuberculosis.

This is not the place to enter upon a discussion with regard to the possibility, or otherwise, of the inheritance of acquired characters; nevertheless, I should like to emphasize the fact that no argument in this paper is based on the supposition of such inheritance. I make a point of this because two months ago, during the discussion on Mr. J. F. Colyer's paper, "The Treatment of Children from a Dental Aspect," I ventured to suggest the explanation of the cause of dental caries which I am going to place before you to-night, and Mr. Colyer said in his reply that such a theory involved the inheritance of acquired characters. On the contrary, if it be true that acquired characters are inherited, the whole argument of my paper is negatived at once.

All variations from the normal can be divided into *genetic variations* and *acquired variations*. Now genetic variations are changes arising in a *race* due to natural variations in the germ-plasm of either the ovum or its fertilizing cell the spermatozoon. In other words, the individual is born with them—they are inborn characters. As examples may be mentioned the difference which exists in the colour of the hair and eyes of different individuals or the occurrence of an extra petal in a flower. Such characters are, as we know, capable of being inherited by the offspring to a certain degree, subject to modification due to ancestral influence. Darwin based his whole theory of evolution by natural selection upon the inheritance of such inborn variations.

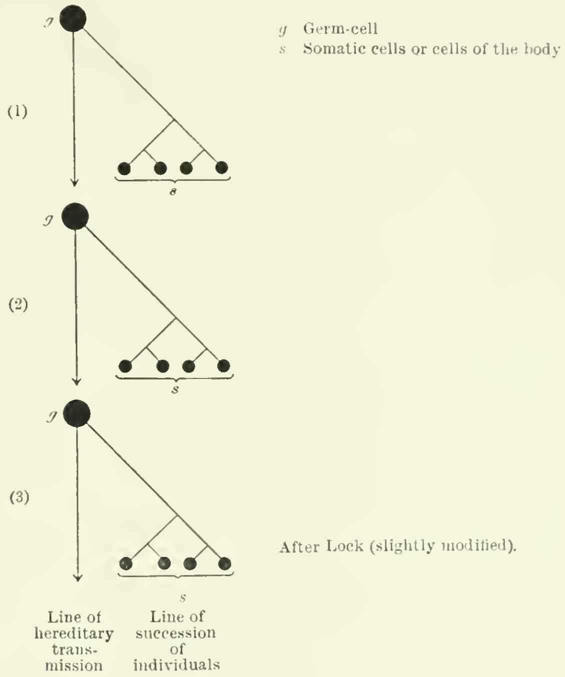
Acquired variations, on the other hand, are of a totally different nature, since they are the changes produced in an individual during life owing to outside influences, as environment, and the effects of use and disuse, &c.—the powerful arm muscles of the blacksmith, for instance, or the power of writing and speaking. Such acquired variations are not inherited by the offspring, at least the whole weight of evidence is against it.

The difference therefore lies, on the one hand, between variations existing in the germ-cells before fertilization, and on the other in variations arising in the somatic cells or cells of the body after conception, during the period of growth of the individual or later, and due to certain definite causes of environment, use, or other comprehensible cause. Weismann, in his well-known work on "The Continuity of the Germ-plasm," puts this very clearly. He says:—

> It is a reversal of the true point of view to regard inheritance as taking place from the body of the parent to that of the child. The child inherits from

the parent *germ-cell*, not from the parent's *body*, and the germ-cell owes its characteristics not to the body which bears it, but to its descent from a pre-existing germ-cell of the same kind. Thus the body is, as it were, an offshoot from the germ-cell. As far as inheritance is concerned the body is merely the carrier of the germ-cells, which are held in trust for coming generations.

This diagram is taken from R. H. Lock's book on "Variation, Heredity, and Evolution:—


g
(1)
s
g
(2)
s
g
(3)
s
g Germ-cell
s Somatic cells or cells of the body
Line of hereditary transmission
Line of succession of individuals

After Lock (slightly modified).

Diagrammatic Explanation of Weismann's Theory.—In this diagram the germ-cells of three successive generations, *g* (1), *g* (2), and *g* (3), are represented, also the individuals, *s* (1), *s* (2), and *s* (3), arising from each germ-cell. It will be seen that each successive germ-cell arises from the germ-cell, *g*, of the parent, and not from the somatic or body cells, *s*; hence variations acquired by the somatic cells, *s* (1), cannot affect the germ-cell, *g* (2), of the next generation, since *g* (2) is descended directly from *g* (1), not from *s* (1).

There are, it is true, certain cases of acquired characters which appear to be inherited, but the whole weight of evidence is so strongly against the possibility of such inheritance that it may be supposed that further inquiries into these cases will lead to an explanation on other grounds. The whole question is one of enormous importance, since if the habits and tendencies of our children are largely determined by the habits and tendencies we ourselves acquire before the age of discretion, the responsibility of the parent is overwhelming. I had not intended going into this question, but Mr. Colyer's contention so invalidated my whole paper that I feel some explanation is necessary.

Now in the same way acquired immunity, such as is conferred upon the individual by, say, an attack of chicken-pox, cannot be hereditary, else had chicken-pox died out long ago; whereas we know that the children of parents who have themselves acquired immunity against the disease by an attack earlier in life are just as liable to contract the disease, when exposed to infection, as the children of parents who have never had it, and who are therefore not immune.

On the other hand, the natural immunity against certain diseases possessed by some races is truly hereditary. Here again, however, it is not the inheritance of acquired characters which confers the immunity—that is, the immunity was not acquired by the parents and handed on to the offspring. This natural immunity does not resemble acquired immunity, in that it is not the outcome of a previous experience of the disease by the individual; it is more a non-susceptibility, to use a clumsy word, to that particular disease, and has arisen in the race simply as a result of the weeding out of the more susceptible individuals, owing to their succumbing to the disease at once. Thus breeding only takes place among the less susceptible individuals, and so gradually the race is rendered immune to that particular disease. This is so well put by Dr. Archdall Reid that I will, with your permission, read you what he has to say on this part of the subject. He says:—

> It is universally admitted that men differ greatly in their susceptibility to infection and in their power of subsequent resistance. In England, for example, hardly anyone escapes measles, whooping-cough, or tuberculosis unless he be immune, or death unless he be resistant. In other parts of the world no one weak against malaria, typhoid fever, or dysentery is able to survive. Whenever any form of selection is stringent it is accompanied by an evolution of those qualities which enables the survivors to escape. It follows, in the case of disease, that "selection should cause an evolution of an inborn power of resisting infection"—that is, inborn immunity—"or of an inborn power of recovering from infection. . . ." When the disease is one against which immunity cannot be acquired the race has undergone an evolution of inborn immunity: thus Europeans, who have suffered severely from tuberculosis for thousands of years, resist infection by it, or when infected recover from it more

easily than African negroes, who have suffered less, and much more easily than American Indians, who until lately had no experience of the disease.

Man's evolution against malaria is more striking and conspicuous than that occasioned by any other disease, and that for two reasons: first, because in many districts infested by its microbes it is so prevalent and virulent that no man resident in them escapes infection unless he is immune, nor death unless he is resistant. The elimination of the unfit, therefore, has been very thorough, and . . . evolution against malaria has been very considerable. Secondly, the illness occasioned by the disease is of a very sudden and marked character, and therefore observers are easily able to contrast its effects on individuals of different races, and to perceive how much more resistant are those races which have had prolonged experience of it than those to which it is strange.

So considerable has evolution against malaria been in various parts of the world that it is scarcely necessary to bring forward evidence in proof of it. Nothing, indeed, can be plainer than that different races of mankind differ vastly in their powers of resisting the disease, and that those races that have had extended and disastrous experience of it are much more resistant than those who have had little or no experience of it.

In Ceylon there died of malarial fever, per 1,000 of the population: Negroes, 1·1; Europeans (English), 24·6.

I think, therefore, that it is fairly evident from the foregoing how certain diseases are kept in check and prevented from exterminating the race by the aid of natural selection. Unfortunately, however, there are certain other diseases which, owing to our artificial civilization, are not susceptible to Nature's action. Of these I will mention only two—insanity and dental caries. The reason that natural selection cannot keep any check on the spread of these is due to the fact that neither incapacitates the sufferer in the vast majority of cases from leaving plentiful offspring.

I will first deal very shortly with insanity, as it affords an almost exact parallel to dental caries, and has the advantage of being under State control. This means that very careful records of any increase or decrease in numbers are kept. Now insanity has increased enormously during recent years, as the figures here will show:—

FIGURES FOR IRELAND.

Year		Ratio of lunatics
1851	... ...	1 in 657
1861	... ...	1 in 411
1871	... ...	1 in 328
1881	... ...	1 in 281
1891	... ...	1 in 222
1901	... ...	1 in 178

The reason for this increase is not far to seek. In olden times the insane were treated like wild beasts and thrown into dungeons, where they seldom survived for many years owing to the terrible treatment they were submitted to. Now, on the other hand, the State takes care of them, houses them, feeds them and clothes them, gives them the kindest treatment and the most skilled medical attendance, so that an enormous majority recover and, leaving the asylum, marry and have children, all or most of whom inherit their parents' predisposition to insanity; for insanity, or rather the particular mental defect which leads to it, is undoubtedly inborn and not acquired, and therefore hereditary. Dr. Reid again on this subject says:—

> Insane persons have multiplied a hundred-fold with civilization to such an extent that . . . while in the United States the population doubled in little more than thirty years, the insane increased sixfold, so that in the last decade the increase in the population was 30 per cent., and that of the insane was 155 per cent.

The following case of Dr. Reid's helps to emphasize what he says:—

> A woman who is more than half a lunatic came to live with two sisters—one a total, one a partial idiot. She married a very dull, partially idiotic man, and had almost immediately to be taken to the asylum. There she gave birth to a complete idiot, and was sent home a few weeks afterwards, with the result that the same thing had been repeated *nine times*.

I will now turn to dental caries itself. I hope to be able to show that the increasing prevalence of this disease is largely due to the same cause as insanity, viz., the removal of the influence of natural selection.

The great prevalence of dental caries among civilized races at the present day has been attributed by Dr. Sim Wallace and many others to the soft and clinging nature of our food. Dr. Wallace, who above all others has drawn attention to this food question, and who has given a great deal of time and thought to it, maintains that it is the cleansing action upon the teeth of their coarse fibrous food which protects the teeth of savages from decay, and, on the other hand, that it is the soft, clinging, and easily fermentable food of modern diets which accounts for the fearful ravages of the disease among civilized men. From Dr. Wallace's writings I gather he does not allow that there is any difference in the relative susceptibility of the teeth of the savage and civilized man, and that the difference in the amount of dental disease found is due solely to change of environment as regards the teeth, that is, the accumulation or otherwise of soft food masses around the teeth. On this hypothesis, therefore, every child all the world over is born with the same relative susceptibility to dental caries.

Now, feasible as this theory is in many ways, I venture to think that it does not account for all the known facts with regard to dental caries. For instance, why is it that there are some people who never show a spot of caries all their lives? These "immunes" have as a rule the same habits, and eat the same food, in the same way, as their brothers and sisters, some of whom may lose all their teeth before they are 50. We see among our patients some with receding gums and all the interstices between the teeth blocked with soft fermenting food, and yet no caries results. Others we see who keep their mouths spotlessly clean, so far as is possible. Their teeth all antagonize perfectly, and there are no spaces between them, yet cavity after cavity appears and in spite of all our own efforts the whole mouth is a wreck before they reach the age of 35. I have two such cases in my mind at the present moment.

I think these facts militate very strongly against the food theory, for given the same degree of susceptibility in everyone, then all who are submitted to identical conditions should suffer in an equal degree.

I will now turn your attention to the point of view from which we have already regarded other diseases, as malaria and insanity. Savage man is free from dental caries for the same reason that he is free from other diseases. We have seen how, in the case of malaria, inborn immunity is produced by natural selection allowing the death of the more susceptible. The same law applies to all undesirable variations which may appear, *and susceptibility to disease—including, of course, dental caries—is only one of the many possible undesirable variations.*

It is unnecessary to go into the detailed working of this law with regard to dental disease especially, for its operation is the same for all diseases against which inborn immunity can be gained. Defective teeth must incapacitate the individual from obtaining sufficient nourishment as rapidly and easily as his fellows, and hence he fails in the struggle for existence. Thus all susceptible individuals are removed from the race by natural selection, as already shown in the case of malaria, with the result that breeding takes place only among those not so susceptible, and so the race as a whole is kept free from the disease.

Among civilized races, however, the conditions are very different. The soft, easily digested food which is eaten requires very little mastication, so that the loss of, say, two molars on each side above and below would not handicap him at all in obtaining sufficient nourishment, as compared with his fellows who possess all their teeth.

Then again the whole aim of dentistry is to patch up defective teeth and render them efficient, and so put the individual on an equal footing with his neighbours. Although seriously defective teeth do undoubtedly often lead to illness, and even very occasionally to death, the cases are certainly very few where they interfere with marriage and the production of a large family.

Here, then, is the parallel to insanity about which I have already spoken. In both cases the action of natural selection in eliminating the unfit, and so perpetuating only the non-susceptible or immune individuals, is prevented by modern science. We have seen that in the case of insanity the gradual increase in the numbers of the insane is due to the recovery of, and breeding by, persons possessing a predisposition to insanity. Now these, even by the latest statistics, do not number much more than 1 in 178; whereas the number of persons suffering from dental caries is certainly not less than 95 per cent. of the population in Europe to-day; in other words, nearly every child is born of susceptible parents, and any retarding influence on the increase of susceptibility which the few immune individuals may exert is quickly lost by the marriage of their children with susceptibles.

Can we, then, wonder at the enormous and increasing prevalence of dental caries at the present time?

In the paper by Mr. Norman Bennett which I have already referred to he mentions some observations by Dr. Black "on the inheritance of a liability to caries, even as regards particular teeth and particular positions." This is absolutely in accord with my ideas, since if the susceptibility to dental caries is hereditary it is extremely probable that the undesirable variation which produces that susceptibility would affect different teeth to a varying extent.

As regards the small proportion of the population who are immune to dental caries, I think their presence may be explained by *reversion*. We know that in any species individuals are occasionally found who revert in one or more characters to an ancestral type. As I have already indicated, "immunity" against any disease must be regarded simply as a variation—a favourable one in this case—and as such is able to be preserved by natural selection. A member of the human species is thus able to revert to this ancestral variation of immunity. In the same way we often meet with reversions to ancestral types in the shape of the jaws, teeth, and ears, and the contour of the head.

Equally intelligible by the light of the study of heredity are such conditions as narrowness of the jaws, leading to the various dental irregularities with which we are so familiar. The reduction in size of the teeth, compared with savages, is, of course, equally the result of the absence of natural selection.

I have attempted in this paper to deal with the causation of dental caries, and to trace it to the absence of natural selection among civilized races. As to whether I have met with any success in my attempt to throw some light on this obscure problem I leave to your decision. I feel, however, that the general opinion of the dental profession, and even more the public, towards this question is, that no theory of the cause of the existing prevalence of dental caries, however true it may be, is of the least value unless it opens up the road to preventative treatment. In a very few words, therefore, I will try and point out what practical conclusion can, I think, be drawn from this study.

First, then, I will repeat the main point of my paper, that the chief cause of the increasing prevalence of dental caries among civilized races lies in the *teeth themselves*, and is due to their increasing susceptibility owing to the removal of the action of natural selection.

This is the main point I wish to emphasize, and I believe that to recognize its truth is of enormous importance, for it follows that so long as civilization progresses and the survival of the unfit continues, the susceptibility of our teeth to dental caries will get greater and greater. This is an unpleasant conclusion, but that does not warrant us in refusing to recognize it ; and if we will not recognize it, or, recognizing it, do not take measures to cope with it, the results will sooner or later be disastrous to coming generations. Now if the main cause lies in the teeth themselves, and not in the oral secretions, or the proportion of lime salts in the blood, or any other outside factor, then we have a fact of really great importance on which to work. We learn, in fact, that all or nearly all teeth are capable of being infected owing to their susceptibility, and the obvious treatment is to prevent infection ; in other words, to adopt the old course, which hitherto has only been followed empirically, of preventing food lodging around the teeth, that is, absolute cleanliness.

Now there are two distinct types of cleanliness : natural cleanliness and artificial cleanliness. Of these natural cleanliness is by far the most desirable, but at the same time the most difficult to attain. It consists chiefly in the regulation of the diet, preference being given to such foods as do not tend to stick around the teeth, and the elimination from our diet, so far as is possible, of soft, sticky and easily fermentable foods, which are not easily washed away by the saliva during mastication.

The work of Dr. Sim Wallace in this direction is so well known that it is quite unnecessary for me to go into further details. Dr. Wallace has experimented with most of the different foods which are usually eaten to-day, and has classified them according to their natural tendencies to adhere to the teeth after eating, also especially naming such articles of diet as have a cleansing action on the teeth during mastication. I think, therefore, the ideas I have brought forward to-night tend to emphasize the importance of much of Dr. Wallace's work. I only differ from him as to the etiology of dental caries. Secondly, as to artificial cleanliness. By this I mean all such artificial methods of cleaning the teeth as toothbrushes, toothpicks, silk, mouth-washes, &c. I think that such aids to cleanliness are absolutely indispensable considering the conditions under which we live. Natural cleanliness is, of course, the ideal, but I am convinced that perfect natural cleanliness is an impossibility for the general public; modern food simply will not permit it. Dr. Wallace does not agree with me here, I know; he places very little reliance on the toothbrush, and none at all on antiseptic washes as preventatives, and he quotes cases which bear out his contention. If I may mention one of these cases, however—that of the little boy he often refers to, and whose models he showed us in March. I strongly suspect that child to be an *immune*. Indeed, I feel pretty sure that if Dr. Wallace fed that child on every undesirable article of food mentioned in his book for a year, his teeth would still remain free from caries.

Valuable, therefore, as is natural cleanliness as a protection against caries, it can only be absolutely efficient among races living in obedience to Nature's laws, that is in a savage state. With civilization and artificial methods of feeding and living comes the need for artificial care of ourselves. The further one departs from Nature the greater the need to find artificial substitutes for the protecting influence we thus prevent her from exercising over us.

To close with the words of Dr. Archdall Reid:—

> Owing to improvements in medical science and the consequent survival of the hitherto unfit, tremendous problems have arisen, the solution of which cannot long be delayed without disaster to the species.

DISCUSSION.

Mr. F. J. BENNETT congratulated the author upon the excellence of his material and the boldness of his ideas. A careful perusal of the data was necessary before one could say anything adequate, but there were one or two points he might mention. The author had alluded to two patients, one in which decay occurred in spite of the most vigorous, careful and conscientious brushing, and another in which no brushing or care whatever was taken, and in which particles of food were allowed to lodge in the crevices amongst the teeth. He should like to know whether the author examined with litmus or any other crucial test the amount of acid in the particles lodged in the crevices. The author had driven home the question of the structure of the teeth, and he wished to know whether that was mere opinion or based upon any chemical or microscopical examination.

Dr. SIM WALLACE said there was a great deal in the paper with which he thoroughly agreed, especially where the author followed Weismann, but where he followed Mr. Archdall Reid he was afraid he could not agree with him. With regard to the table of ratios of lunatics, showing the increase of lunacy supposed to be due, he presumed, to interference with natural selection, the table covered about two generations, and if the figures were right the increase in lunacy was alarming. But the causes of lunacy were well known to the dental profession. Mothers had particularly bad teeth and oral sepsis; they could not masticate properly and were absorbing and swallowing poisons. They were often so badly nourished themselves that they could not nourish their children: the children were fed on artificial substitutes for mother's milk and later on on pap. In other words, the great increase of lunacy was concomitant with and greatly dependent upon the great increase of malnutrition: it was an acquired characteristic. One might in the course of a thousand years be able to appreciate some very slight difference in genetic progression, but it would be difficult even to say that. If lunacy was increasing on account of the abeyance of natural selection, why was not bronchitis, typhoid fever, typhus, and tuberculosis actually progressing? Why was tuberculosis decreasing amongst white men? Why was scarlet fever and a whole host of other diseases not progressing in a similar way to lunacy? In the first place, however, it would be well to consider whether it was true that in uncivilized communities there was relatively a great amount of weeding out of those with defective teeth. Take, for example, the Esquimaux or the Pygmies of Central Africa; they had especially good teeth and required to use them. Was there much extermination of those races on account of decaying teeth and septic mouths? He thought not, because decayed teeth and septic mouths were rarities, and when decay was present it was present in such a limited degree that it was doubtful whether any appreciable harm resulted. Then the author went on to say that in civilized countries the law of the survival of the fittest was practically nullified and that the teeth became susceptible to dental caries. He could not see that the law was nullified, and Mr. Karl Pearson could not see that the law was nullified.

although its effect might be postponed for a time. Decaying and decayed teeth, oral sepsis, and concomitant and consequent diseases tended to produce unfitness, and unfitness neither tended to long life nor early marriage. Mr. Archdall Reid said that alcoholism tended to bring about sobriety in the race by the elimination of those most predisposed to drunkenness; but Professor Osler had said that if he were asked to say whether more physical deterioration were produced by alcoholism or defective teeth, he should unhesitatingly say defective teeth, and that was the idea of dentists in general. Alcoholism was a disease of adult life, while dental caries was a disease which started in early childhood. Unfortunately those who took to alcoholism generally took to it later in life—sometimes after marriage—and they often left large families; but the unfortunate children who suffered most from dental caries and concomitant malnutrition and consequent ill-health were specially predisposed to diseases which carried them off before they had reached maturity, or at least which might prevent them entering into matrimony. Surely, then, those who were most predisposed to caries must have less offspring than those who had the most excellent teeth and concomitant fitness. Moreover evolution took place not only on account of the extermination of the unfit, but also by the predominance of the prepotent, and it was general health and fitness for the environment that produced prepotency. If during a lifetime an animal had been well nourished and free from disease, and if that animal was mated to another which had been relatively ill-nourished and subject to disease, then, other things being equal, there would be a tendency for the prepotent animal to leave its type, rather than the animal which was not healthy, strong and vigorous: for ill-health, whether arising from defective teeth or otherwise, gave rise to constitutional symptoms, affected the blood and the consequent nutrition of the germ-cells; and if the germ-cells were slightly injured by starvation, toxins, poisons, or any other noxious or unphysiological agency, there would be a tendency to relative impotency of the determinants or whatever they might call the physical basis of the potentialities of the germ-cells. As the factor of the tendency to prepotency of the fittest had been overlooked, and as it was an important factor in evolution, he might perhaps be allowed to dwell on it more fully. These who had bred stock and attempted to keep to the artificial and unphysiological standard would know how difficult it was even to maintain the most artificial, the prize standards. He once tried to breed fancy varieties of pigeons, but after a few years of disappointment he came to like the common blue rock type best, because all deviations from it led to weakness, impotence, and disease. There were, for example, crop and leg troubles with the pouters, eye and wattle troubles with the barbs and carriers, and nesting, feeding, and rearing troubles with them all. The nearer one got to the prize types, *i.e.*, the further one got away from the common standard physiological type, the more unhealthy and sterile the birds became. If a so-called first-class bird was mated to a so-called second-class bird the offspring generally followed the second-class parent, because the second-class parent was the more physiologically perfect, the strongest, the prepotent. Moreover the strain to which the second-class bird belonged was, as a rule, less strained or, in other words, more physiological. Many other illustrations might be given, and if anyone would look into the literature of the subject he thought a great deal would suggest the prepotency of the fittest. Even although there were but few facts to indicate its truth, surely it was obvious that if by any arrangement an animal could follow the fittest parent it would be an immense aid to evolution along satisfactory lines? Surely, too, one could see in this the end of the feud as to whether acquired characteristics were transmitted or not? It was obvious that animals that had variations tending to diseases would, *cæteris paribus*, not tend to have prepotent germ-cells. On the other hand, any animal having variations tending to increased general health would tend to have prepotent germ-cells. In other words, any useful variation would tend to increase until it ceased to be useful, while any useless or harmful variation would tend to disappear so long as it was occasionally the origin of disease or unfitness of any kind, even although it might not involve premature death. He did not wish it to be supposed that he meant that prepotency was not as a rule inherited. No doubt if favoured by healthy nutrition it tended to persist hereditarily. Indeed, such nutrition would appear to favour the doubling of the constituent parts of the determinants at the expense of the weak or injured constituent parts which had been derived from the weak or unhealthy parent. When prepotency became established in that way it simply tended to run on indefinitely or until counteracting forces reversed it, but it would surely be a frightful stretch of imagination to argue that special fitness resulted from dental caries, septic roots, septic mouths, and concomitant and consequent diseases.

Mr. NORMAN BENNETT thought the author was to be congratulated upon dealing with a very difficult subject: the application of natural selection to artificial conditions. The author appeared to trace the onset of dental caries to an absence of immunity derived from genetic variation. Primitive races were liable to certain diseases—measles, for instance—in a very exaggerated form, while civilized communities had them only in a modified form. Dental caries was on rather a different footing. Palæolithic and neolithic man had teeth that were practically free from caries, which was a different thing from an immunity derived from the survival of those who were most fit by genetic variation. The author appeared to think that primitive man had immunity from a disease which was not known to exist at that time. Dental caries was essentially, it would seem, a disease of modern times, and it was hardly fair to assume that primitive man was immune from a disease that did not exist. He did not see that it was quite fair to draw an analogy between diseases which killed and diseases which did not kill; the disease which killed or rendered incapable obviously reduced the individual to such a condition that he was not likely to propagate the species, but a small amount of dental caries was not likely to leave the individual less able to propagate his species: in other words, there was no reason in the nature of things why primitive man should not have had dental caries, and it was not to be imagined that one or two small carious cavities in the molars would have rendered a neolithic man less likely to propagate his species than a man without them. Although that line of reasoning might tend to show that primitive man might be protected against dental caries in an extreme degree in a way that civilized man would not be, it was not fair to push it to such an extreme as to argue that he would not be affected by it at all. If the author was correct in thinking that the seat of immunity was in the teeth themselves, why was dental caries essentially a disease of childhood? Assuming that acquired immunity existed in some other part of the organism—such, for instance, as the fluids of the mouth—it would be conceivable that such an acquired immunity might exist for a particular period, but not if it was assumed it was in the teeth themselves. With regard to the lunacy statistics, they included all forms of mental incapacity: the vast majority in the asylums were affected with general paralysis of the insane, and that very much invalidated the value of the statistics. If the statistics were derived from forms of lunacy generally supposed to be inherited, there might be something more in them, although even then it would have to be considered that within comparatively recent times there had been great changes in the conditions of life—that the stress and strain of artificial life must have produced a great tendency towards the increase of lunacy.

Mr. KENNETH GOADBY thought the author had brought forward a syllogism in which his middle term was undistributed. The question was: Is dental caries a disease? Diseases were generally regarded as having some sort of pathological reaction on the tissue affected, and showing some sort of attempt on the part of the organism to throw it off. That could not be said of dental caries. With regard to the effect of disease upon offspring, he was rather inclined to join issue with the author. It had been definitely shown that an animal was capable of being immunized to a disease and bringing forth offspring that were immune to that disease. Such offspring would resist thirty or forty times a fatal dose of a given organism to which the mother was immunized. It was very difficult to confine oneself to a general consideration of the question without any reference to its local pathological signs, and very difficult to leave on one side the question of chemical or bacteriological local causes. Since Miller's work dental caries had been looked upon as a purely local mechanical process, in which there was no question of disease but merely a question of more or less mechanical destruction of a physico-chemical nature, in which bacteria acted as agents. That there was some sort of general oral condition in which natural selection played a part was quite possible, but he did not agree that in the teeth themselves must be found the immunity to dental caries. It was a fallacy to say that the West African negroes were immune to malaria. If children living anywhere near the malarial areas were examined it would be found they had large spleens and that their blood contained malarial parasites. The weakly ones died, and a certain amount of fresh, acquired immunity was set up in each case; but if negroes who had acquired immunity in the one district were taken to another malarious district, as the West Coast negroes were taken on an expedition to the Soudan, it would be found they were not immune. In the Soudan, in a recent expedition, the West African negroes were in a district for twelve days, and when the main contingent came back they found 150 out of 250 men were down with malaria. They were the so-called immunes, but they had had an extra big dose and were quite as ill as the white officers. Resistance to disease was undoubtedly in many cases produced by the infection of people to such a disease and the gradual elimination of susceptible persons, and to acquired immunity of such persons. It has been experimentally shown by Bulloch and others that immunity could be given to the offspring by immunizing the mother before birth.

Mr. H. BALDWIN thought both ideas should be accepted, but in their proper proportions. It seemed to him the great cause of dental caries was the fact that teeth were dirty with micro-organisms, but Dr. Sim Wallace would no doubt agree that some teeth by their shape were more likely to remain dirty than others, and teeth of a non-self-cleansing shape were likely to be inherited, as were teeth of imperfect construction. Teeth varied very much in structure, and particular characteristics of structure might be hereditary, and therefore affected the question to some extent. He was, however, fully convinced that the main cause of caries was what Dr. Sim Wallace always insisted upon, diet. Some years ago he attended a Tamil patient who used to spend a year in England and a year in India alternately. Before she came to England she had perfect teeth, and all her relatives in India had perfect teeth, but when she came to England she had caries. In India she said she had no caries or any trouble at all, but after six months residence in England she invariably developed carious cavities and came regularly to him to have them filled. Evidently the kind of feeding which she had in England was the cause.

Mr. F. J. BENNETT said he had referred to testing the cavities, where one cavity was immune although food was lodging in it, and the other was decayed although it was carefully brushed. The question was whether any acid lodged in those spaces.

Mr. STANLEY MUMMERY, in reply to Mr. F. J. Bennett, said he had not tested the saliva of the patients, but he thought the reaction of saliva was undoubtedly a predisposing cause, and one to be taken into consideration. With regard to the structure of the teeth, Dr. Miller had published the results of his investigations, and had pointed out that, although the structure of teeth might not vary analytically, the molecular structure might vary immensely. Dr. Sim Wallace seemed to think that the large increase of insanity was to a great extent due to the absence of breast feeding and consequent malnutrition. That might be so to a certain extent, but he thought there was less reason to suppose that it was due to this cause than that it was due to the absence of natural selection. With regard to tuberculosis decreasing, that was a very doubtful point. Since the open-air treatment had come into vogue a large number of cases had been cured which before would have died, and that had to be taken into account.

Mr. KENNETH GOADBY thought it was generally accepted that tuberculosis was decreasing both in virulence and extent, quite apart from sanatoria and open-air treatment.

Mr. MUMMERY referred to Dr. Sim Wallace's remarks with regard to prepotency, and said that Dr. Burbank in America had done a great deal of experimental work in producing new varieties of plants, wheats and potatoes; and the results did not seem to bear out Dr. Wallace's statement with regard to variation from the normal standard tending to produce offspring more susceptible to diseases and to general weakness. By careful crossing, potatoes had been obtained immune from the potato disease. With regard to dental caries in primitive man, he had examined a good many skulls and a fairly large proportion had carious teeth. He did not think it was quite a fair argument to say that teeth could not be immune from a disease that did not exist. Undesirable variations such as a tendency to disease were certainly kept down by the agency of natural selection. With regard to dental disease being one of childhood, that was very largely because children during their schooldays took practically no care of their teeth. Later on they began to learn the importance of the toothbrush and that delayed the course of the disease. It was rather difficult to answer the question as to whether dental caries was a disease. In any case it did not affect the question at issue, since he had regarded dental caries not as a disease but as an undesirable variation. With regard to the inheritance by the offspring of their mother's immunity, he could not agree with Mr. Goadby, as he believed the immunity in these cases to be acquired from the mother *in utero*. If acquired immunity could be inherited, how was it that chicken-pox, whooping-cough and measles had not died out? As to negroes not being immune to malaria, an enormous number *did* die of malaria, and very likely as many negroes became infected as Europeans, but they certainly did not die in such great numbers. With reference to the shape of teeth, he thought that had a very great effect on the amount of caries. If the teeth were of a peculiar shape, with very bulbous crowns and narrow necks, food tended to stick round them, and more caries naturally resulted.

Odontological Section.

June 22, 1908.

Mr. J. HOWARD MUMMERY, President of the Section, in the Chair.

A Case of Diffuse Fibromata of the Gums.

By ARTHUR EVANS, M.S.

A FEMALE patient, aged 19, was admitted into Westminster Hospital in March, 1904, suffering with enlargement of the posterior ends of the alveolar margins. She noticed the swellings beginning in the early part of 1903, when the upper and lower posterior ends on the right side only were affected. In November of that year a tooth was extracted from the upper and one from the lower jaw, but this did not improve the condition, and the swellings continued to increase in size. Soon after the posterior ends on the left side began to enlarge. In addition to this there was noticeable on admission a thickening of the buccal mucosa, which extended upwards from the right posterior alveolar end to the soft palate, running in the anterior pillar of the fauces and forming a tongue-like process with distinct margins.

The patient had been sent up from the country to seek relief from the pain she suffered during mastication, which was so marked that she had been obliged to dispense with solid foods. The swelling from the upper jaw on the right side was first removed, and a week later that on the left side. This latter growth is seen in the photograph, which is taken from the specimen now in the Westminster Hospital Museum, and it is seen to consist of translucent strands of fibrous tissue, embedded at the base of which is a wisdom tooth.

In January, 1908, the patient returned to the hospital, asking that another operation might be performed, as she had obtained so much comfort from the previous one. The condition of her jaws at this time is shown in the casts taken by Mr. Ernest Gardner,

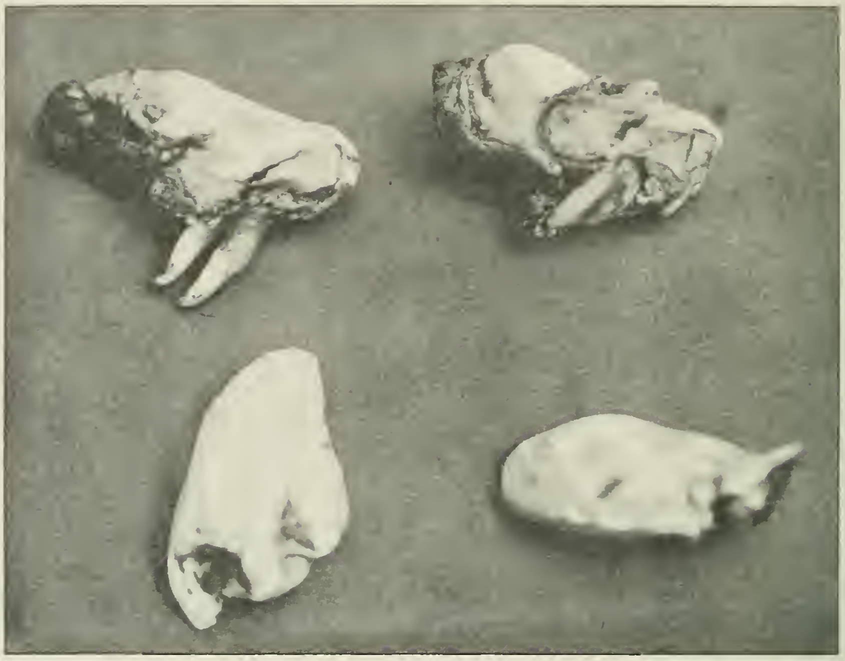


FIG. 1.

Four pieces of hypertrophied gum removed four years after the first piece. The upper two have each a tooth in them. (Slightly enlarged.

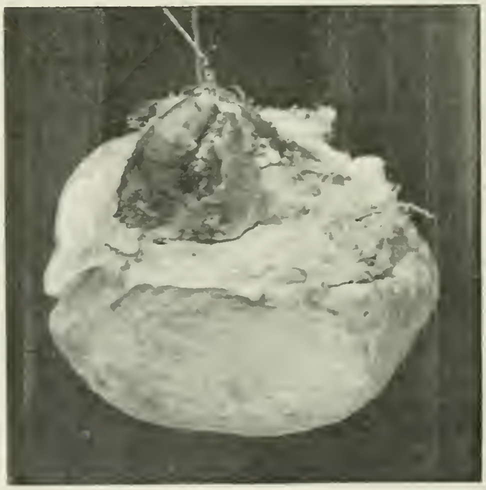


FIG. 2.

Hypertrophied gum with unerupted wisdom tooth removed from upper jaw in 1903. The specimen is suspended by the tooth roots. (Slightly enlarged.)

and which are now in the Museum of the Odontological Section of the Royal Society of Medicine.

The specimens produced are those removed by the second operation. A similar case is reported by Mr. Christopher Heath in the *British Medical Journal*, May 1, 1897, under the heading of "Hypertrophy of [the Gums." The condition had lasted four years and recommenced three months after the removal. In this case the alveolar margins were so wide that they tended to meet in the mid-line, and at first sight suggested a cleft-palate.

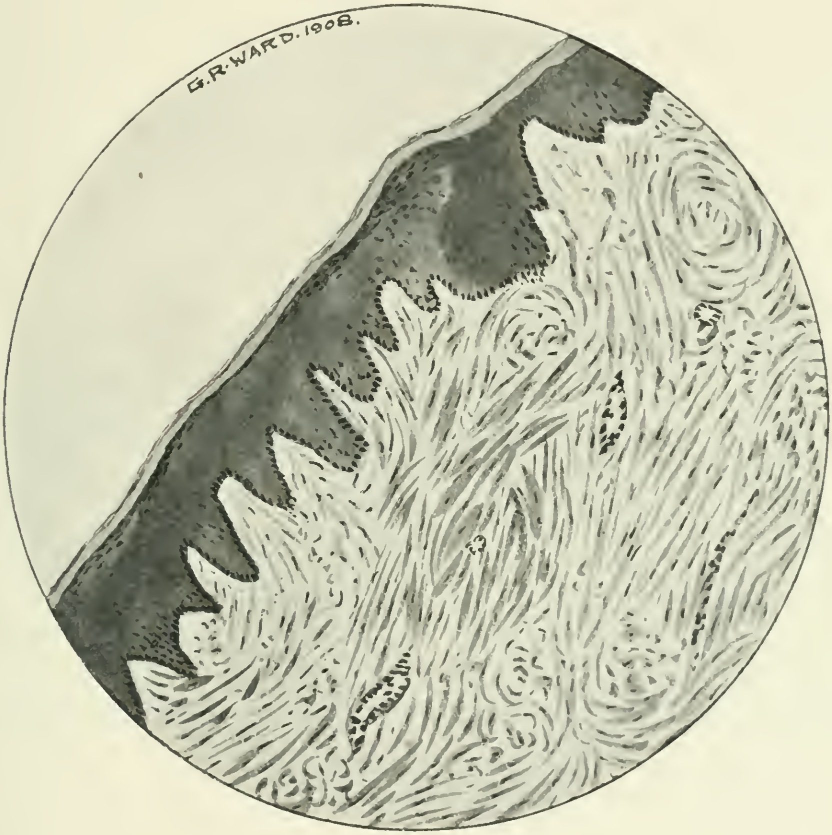


FIG. 3.

Section of first tumour removed (*see* fig. 1). (× 40.)

Report by Dr. Julius Bernstein.—The specimens removed consist of four pieces of gum. The upper two surround respectively a bicuspid tooth, which is deeply embedded in it (the roots alone being visible), and a molar tooth, which is not entirely embedded. The lower two masses show only the sockets of two teeth which are absent. Macroscopically these pieces are similar to the earlier growth, being white and glistening on section, and covered with smooth mucous membrane. Histologically they are composed of adult fibrous tissue arranged in strands, between which is some badly staining homogeneous material denoting some myxomatous degeneration; in places are some thickened arterioles, and around these some round-celled infiltration; the overlying mucosa is slightly thickened, and beneath the thickened papillæ is some excess of round-cells of inflammatory origin. The growth is therefore a fibroma undergoing early myxomatous degeneration. The first growth removed in 1904 was a pure fibroma, and did not show this degeneration.

DISCUSSION.

Mr. BALDWIN inquired if the sockets of the teeth had been removed by the growth. He noticed that some of the teeth had come out very clean. He should like to ask if any force was required to extract them, or if in some way the bone had been removed around the roots of the teeth.

Dr. A. W. BAKER said he had had a similar case some years ago, in which the growth appeared to be associated with epilepsy. The patient also suffered extremely from excessive salivation. He should like to know if either of those conditions were present in Dr. Evans's case.

Mr. J. F. COLYER said the case seemed to him to be one of hypertrophy of the gum, and the interesting feature to him was that it was mainly limited to the molar region. About fifteen years ago he had two patients, boys, with a general hypertrophy of the gums right away round. Heath maintained that the condition could not be got rid of unless not only the hypertrophied gum, but also the margin of the alveolar process, was removed. He took the patients to Mr. Boyd, who simply pared off the hypertrophied gum. He saw the patients about eight years afterwards, and although there had been a certain amount of recurrence, it was not marked. But the interesting feature was that one boy had kept his mouth clean while the other had not, and while in the first case there was comparatively no inflammatory process, in the second case there was distinct development of granulation tissues in the pocket between the teeth and the gum.

Mr. EVANS, in reply to Mr. Baldwin, could not say the amount of force which would have been necessary to make an extraction. He thought that the fibrous tissue had almost lifted the tooth out of its socket. When he made an incision right down to the bony structures the piece of altered gum was easily removable and brought the tooth with it; there was no question of extraction.

In his case there was no connection with epilepsy. With regard to Mr. Colyer's point about keeping the teeth clean, he believed that failure in this matter had nothing to do with his own patient; she had taken great care of her mouth and there was nothing of the inflammatory type about it. The fibroid was quite a slow growing one.

Supernumerary Teeth.

By R. McKay, M.R.C.S., L.D.S.

This case is one of supernumerary teeth preceding and preventing the eruption of the maxillary central incisors. When aged 11½ the permanent dentition was normal with the exception of the absence of the maxillary central incisors and the presence of a cusped supernumerary tooth in the position of the maxillary right central incisor (fig. 1). Skiagraphy (fig. 2) showed the presence of two supernumerary teeth with the two permanent incisors above. Both supernumerary teeth were removed (fig. 3) and the centrals have taken two years to assume their present position (fig. 4).

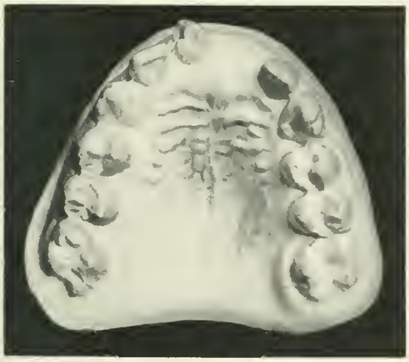


Fig. 1.

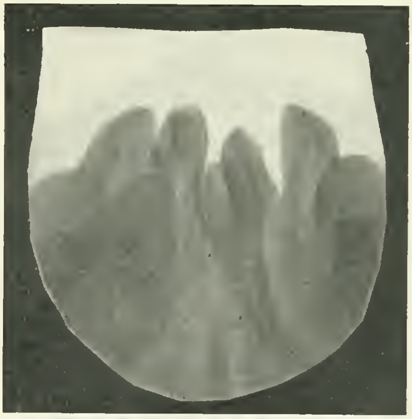


Fig. 2.

The patient, a boy, is the subject of a congenital heart, said by his father, a medical man, to have a patent foramen ovale, but this has never caused him any inconvenience. A cousin has the same heart condition, otherwise there is nothing of note in the family history. The case is an example of a well-recognized group of abnormalities, and is of interest in that, but for the help of the X-rays, the left supernumerary would probably have been allowed to remain when erupted in mistake for a malformed permanent tooth.

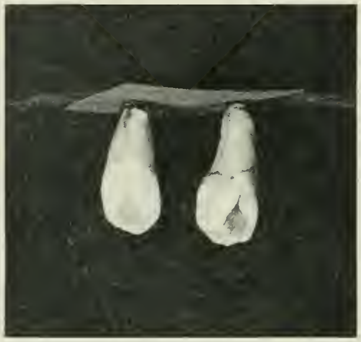


FIG. 3.

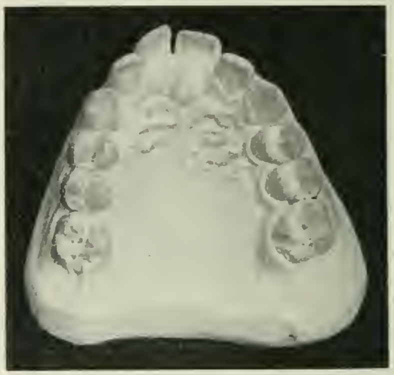


FIG. 4.

A Difficult Extraction.

By ARTHUR W. W. BAKER, M.D., L.D.S.

AT the latter end of March in the present year (1908) Mr. C., aged about 60, a retired Indian civil servant, an active, healthy-looking man, consulted me with reference to some trouble at the left side of his lower jaw, and gave me the following history: He had had for some time back uneasy sensations about the angle of the jaw on the left side, which occasionally became more severe and were accompanied by trismus. He had previously consulted another practitioner, who diagnosed an impacted wisdom tooth. An unsuccessful attempt was made to remove the tooth without an anæsthetic; the patient declined further treatment, and was advised by a mutual medical friend to consult me.

On examination I found that the first and second molars had been removed, evidently some time previously judging from the amount of absorption of the alveolus; these teeth, as well as the molars on the right side, were replaced by a vulcanite denture. There was a small opening in the gum corresponding to the junction of the ramus with the body of the jaw, where, on passing in a fine probe, I was able to touch enamel. A skiagram which had been taken for the patient by Dr. Haughton showed that the tooth was deeply embedded in the bone, with the masticating surface just exposed. It was not lying horizontally, as is frequently the case; in fact, its position was fairly normal. Here, as in some other cases which I have recorded, there was no mechanical obstruction, but the tooth failed to erupt and was causing trouble in its attempts to reach the surface. I plugged the opening in the gum with cotton, so as to get a view of the tooth if possible, and arranged to operate the next day but one.

As I thought it likely the operation would prove difficult, I decided on ether for the anæsthetic, and had the advantage of the assistance of my colleagues, Dr. Maxwell and Mr. Sheppard. For somewhat over an hour, with various elevators and forceps, I endeavoured to effect the removal of this tooth from its bony socket, my chief difficulties being: (1) That the tooth was almost entirely surrounded by dense bone—not the ordinary alveolus—that rendered it almost impossible to introduce an instrument between the tooth and the walls of its crypt; (2) the close attachment of the muscles and soft parts to the angle of the jaw, so that I could not use the engine or chisel to remove the overlying bone without more risk to my patient than was desirable; (3) the very small size of the orifice of the patient's mouth, rendering it anything but easy to apply an instrument so as to grasp the tooth: and lastly, the fact that when the patient was deeply anæsthetized any pressure on the lower jaw at once stopped respiration, so that before I could operate it was necessary to allow him partially to recover. However, just as I was beginning to despair of success I managed to get a forceps sufficiently far down to grasp the tooth and dislodge it. A couple of hours later, when the patient had got over the effects of the ether, I washed out the socket with hot creolin and dusted it well with iodoform, the latter drug seeming to keep a wound in the mouth cleaner than anything else I know of. In order to relieve the pain and swelling which usually follow such a severe extraction I bandaged up his jaw with a thick layer of hot antiphlogistine on cotton wool, ordering its renewal every two or three hours. This, the patient assured me, gave him the greatest relief—in fact, he used to look forward to his "mud" poultices as procuring him immunity from pain. Some forty-eight hours after his operation

I was much gratified to find the wound in the mouth free from sloughs and the tissues about the angle of the jaw quite soft and scarcely swollen. The subsequent clinical history, I find from my notes, calls for little comment. There was some slight swelling along the outer side of the jaw, as the wound granulated nicely, and although a piece of bare bone appeared in the centre of the wound its colour was healthy and justified my opinion that it would not necrose by becoming speedily covered with granulations.

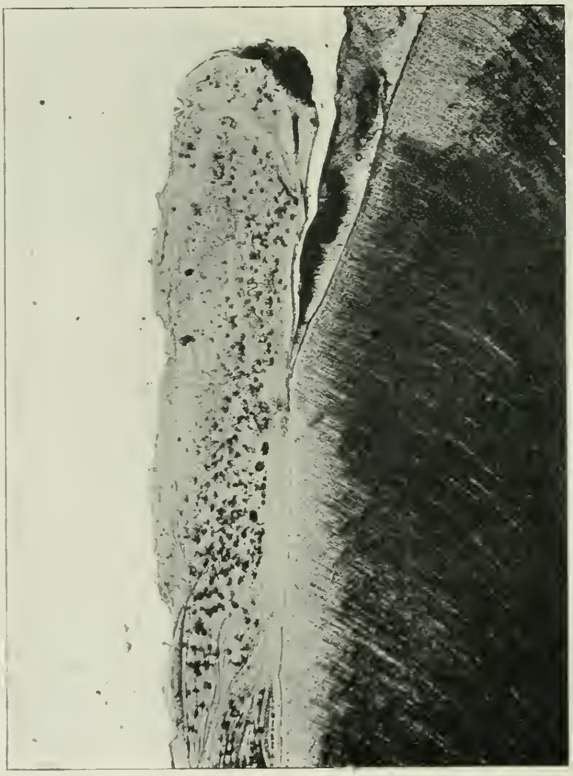


Fig. 1.

Hyperplasic cementum external to the enamel. Longitudinal section. × 50.

On examining the tooth, which was of a yellowish colour, I found that it was $\frac{9}{16}$ in. long, $\frac{7}{16}$ in. wide, and $\frac{6}{16}$ in. thick. Short, thick, and irregularly quadrate in shape, the enamel was very poorly formed, rocky, and evidently in only a thin layer over the crown; it also did not extend quite to the neck of the tooth. This appearance was explained by the subsequent microscopic examination, which showed the enamel to be overlapped by the cementum (fig. 2). The cusps were not formed. There was considerable hyperplasia of the cementum, with several large channels for vessels passing inwards from the surface. The roots, if at any period separate, were now united in a mass of cementum; in fact, the tooth suggested an approach to an odontome, and might best be described as belonging to the class denominated "warty" by Salter. I prepared three ground sections, cutting one transversely close to the apex of the roots; the other two

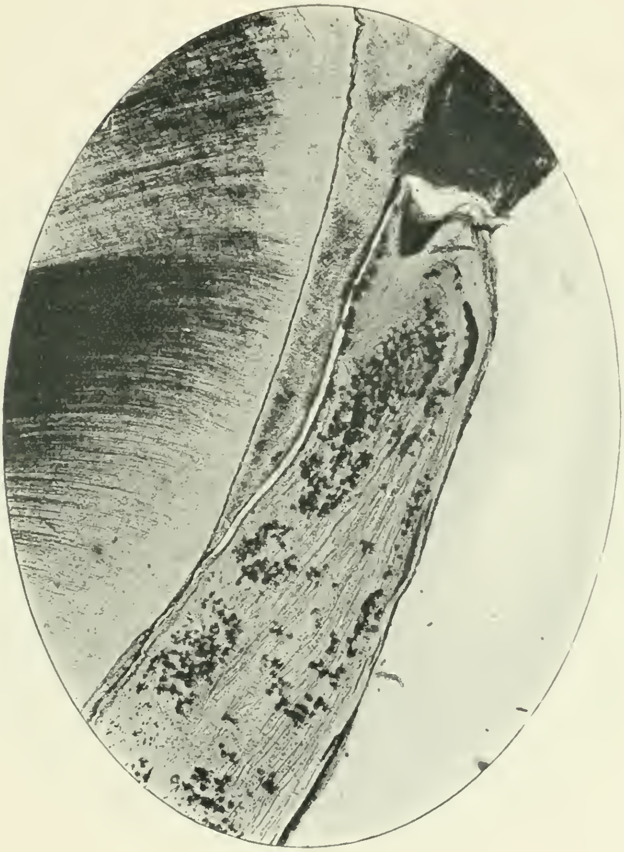


Fig. 2.

Hyperplasic cementum external to the enamel. Longitudinal section. × 50.

were longitudinal through the crown and upper portion of the roots. These sections were placed in alcohol and fuchsin, any soft or imperfectly calcified portions taking up the stain readily. On examining the transverse section, which was taken near the apex of the roots, I found that there was considerable hypertrophy of the cementum, which showed lacunæ and well-marked incremental lines, as well as large canals for blood-vessels. The cementum had completely covered and united the two roots. At one side of one of the roots the cementum and dentine have evidently been absorbed right into the pulp canal. Some of the soft tissue, in a semi-calcified condition, may be observed

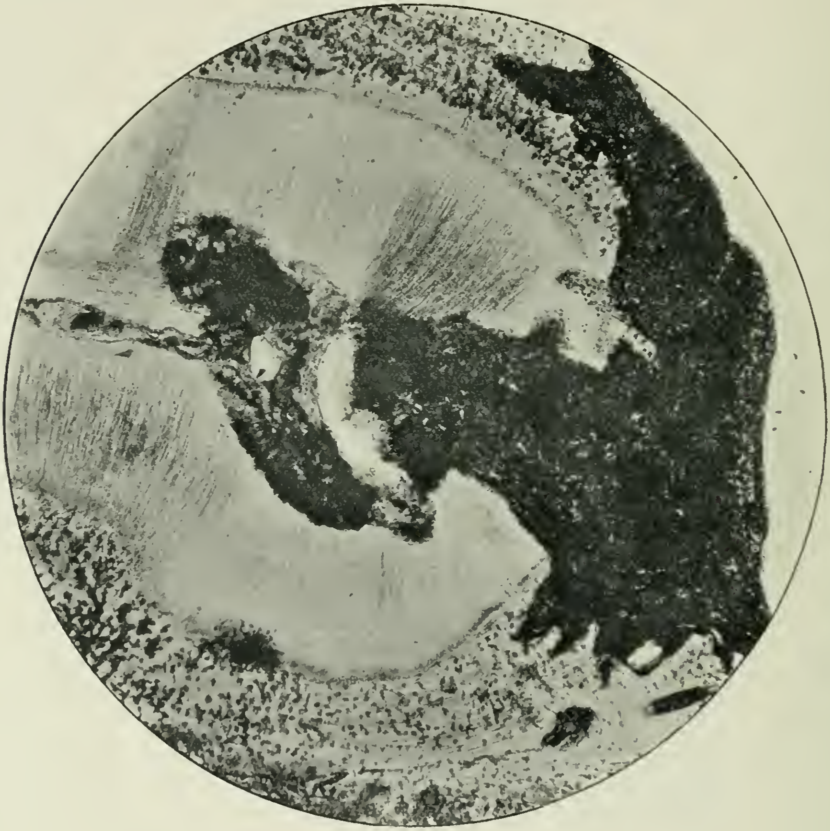


Fig. 3.

External absorption of dentine and hyperplasic cementum; deposition of compact bone. Transverse section. × 50.

stained red, but the greater portion of the invading material, presumably giant-cells, has undergone transformation into bone (fig. 3). The baying out of the cementum and dentine, showing Howship's lacunæ, would tend to prove that this bone was a pathological change rather than a developmental error. As I had not observed such a condition before, I concluded that it was rather rare, and on turning to Mr. Hopewell-Smith's excellent work on dental pathology I found

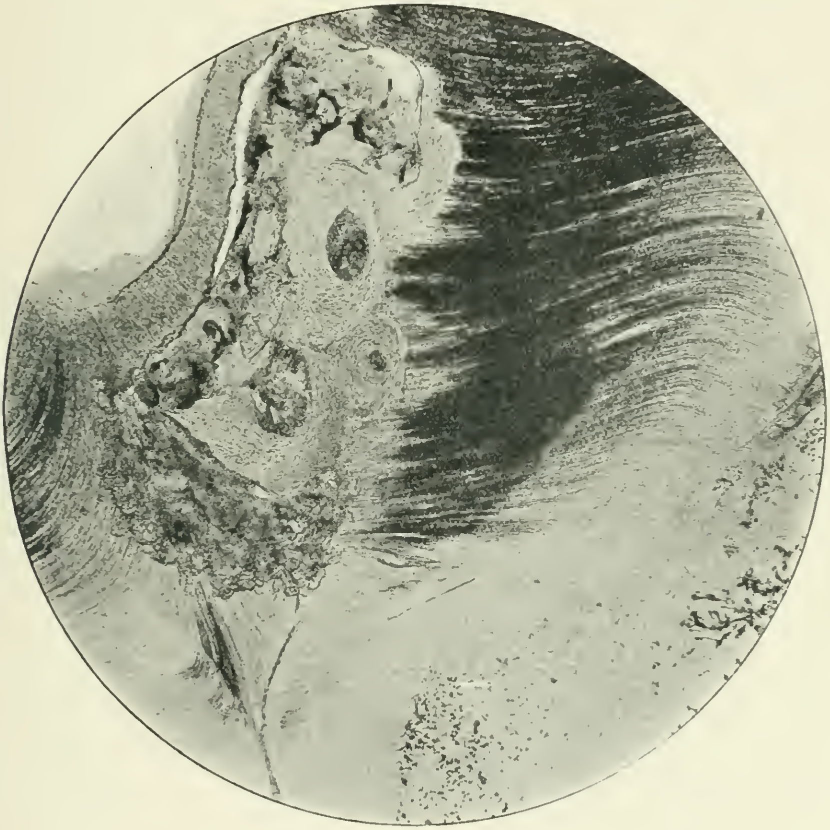


Fig. 4.

Absorption of dentine from internal surface; deposition of compact bone; hyperplasic cementum externally. Transverse section. × 60.

that there were only four other cases on record. I may add that this growth of bone could not be attributed to ankylosis with the socket, as for various reasons it could not have occurred here, this pathological change being most likely due to very chronic inflammation. The longitudinal sections exhibit the dentine fairly regularly developed, but on the whole imperfectly calcified, with evidences of interglobular spaces. The pulp chamber shows that the pulp is undergoing calcification, and is depositing a new layer of calcified tissue as if to protect itself from the bony invasion which is advancing towards it;

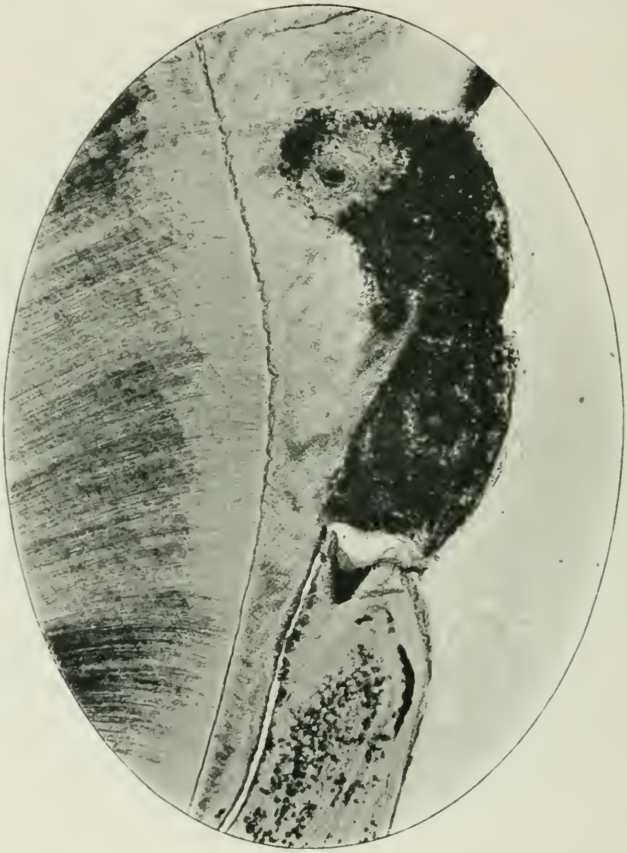


Fig. 5.

External absorption of enamel and deposition of compact bone; hyperplasic cementum external to the enamel. Longitudinal section. × 50.

this bone is similar to that near the apex of the root, and with which it is probably continuous. The enamel is decidedly imperfectly formed and shows but slight indications of Retzius' striæ and Schreger's lines;

enamel spindles may be observed here and there. But where on the same side as the absorption and deposition of bone in the cementum and dentine occurred a like process has taken place in the enamel. This is extremely rare, and according to Hopewell-Smith there is only

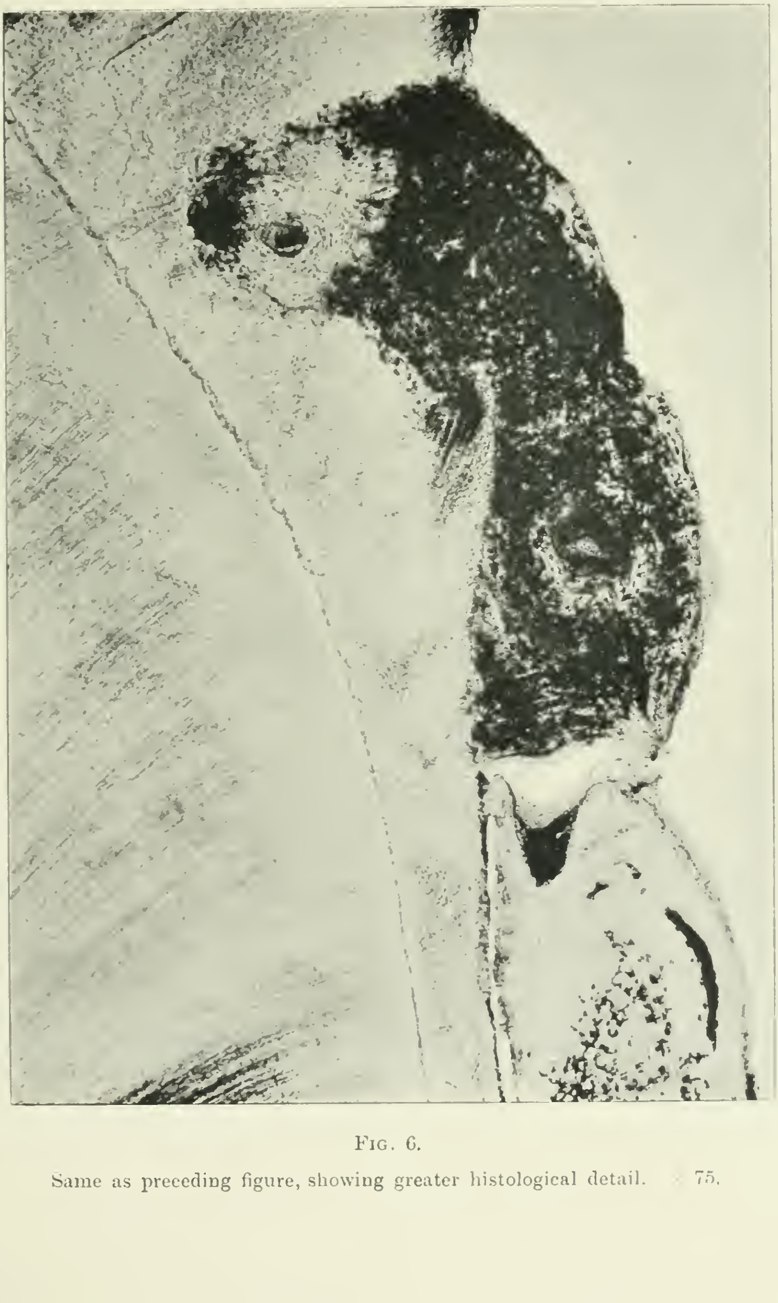


FIG. 6.

Same as preceding figure, showing greater histological detail. × 75.

one other case on record. A thick layer of cementum overlaps the termination of the enamel, which on one side of the tooth is separated from the enamel, apparently by a canal (fig. 2). At the termination of the enamel of the crown, and separated from it by an interval, there is a further deposit of enamel in a thin layer, under the thick layer of cementum. According to the late Professor Miller (as quoted by Hopewell-Smith) the investigations of Hertwig, von Brunn, and Röse led to the conclusion "that the primary function of the enamel organ, for which the name 'epithelial sheath' has been proposed, is not to produce enamel but to give form to the tooth, and that normal dentine is formed only on the inner wall of an epithelial mantle. As soon as the epithelial sheath ceases to grow, then the formation of tubulo-dentine ceases, osteo-dentine, cementum, or bone taking its place. The roots of the human teeth are accordingly formed within a sheath or mantle of epithelium. That this sheath should, under certain conditions, extend its function of forming enamel beyond the normal limit is not a matter of great surprise. We see evidences of this in enamel nodules found on the roots of molars." The above remarks will probably explain the existence of an accessory layer of enamel in my case. In conclusion, I trust that the pathological interest of this tooth may in some measure atone for inflicting on you the details of a troublesome operation.

The accompanying photomicrographs have been taken by Mr. Hopewell-Smith.

DISCUSSION.

The PRESIDENT (Mr. Howard Mummery) asked Dr. Baker whether he thought he was quite justified in using the expression "odontome"—the tooth was more like a normal one. It had undergone a pathological change. He remembered the late Mr. Ackery showing the Section a specimen of the invasion of bone into the dentine. Of course it did not take the place of any tooth, but it was apparently the wisdom tooth, and the changes had taken place, he thought, pathologically.

Mr. MONTAGU HOPSON said a few years ago he had a very similar case both with regard to the difficulty of the extraction and also in regard to the external contour of the particular tooth. It was almost quadrilateral; it was rather imperfectly developed and, furthermore, at what were the apices of the roots there was situated a very deep groove which corresponded with the position of the mandibular nerve. He remembered the extraction quite well. He was at the case for twenty minutes, under ether, and absolutely failed to remove the tooth. He got his senior colleague to take the case into the hospital, and his colleague spent an hour on it, with a very similar experience to Dr. Baker's, and also with a successful result. The tooth was in his possession, and after listening to Dr. Baker's paper he would examine it microscopically and see if it corresponded with the pathological conditions of the case exhibited. He should like to confirm some observations which Dr. Baker had published a short time since with regard to pain which was associated with wisdom teeth which were buried but which were not impacted. He thought there could be no doubt with regard to that. He had in his mind a hospital case which was under his care about a month ago. The patient was an old woman aged 68. Her lower jaw was apparently edentulous, but she had all the symptoms of an impacted wisdom. With a sharp probe thrust into the position where he thought the wisdom tooth might be, he fancied that he struck enamel, and he sent the case to be skiagraphed. The skiagram was an excellent one, and showed the position of the tooth perfectly well. He first of all made an incision down on to the surface of the tooth and then had the gum packed away, and the result was that the following week he was able to outline the surface of the tooth quite well. There was no difficulty in extraction at all. As was frequently found, the teeth in that situation, when wedged into the extreme angle of the jaw, were better seized with a pair of upper forceps than with an ordinary pair of lower forceps. The interesting thing about the case was a fact which possibly explained some of the pain which the patient experienced: that when the tooth was removed he found it was perforated—there was a hole running through the roots, through which also the mandibular nerve passed. The result was that the nerve was ruptured and there was the loss of sensation in the half of the lower lip on that side. He had only seen the patient once since and the sensation had not then returned, but he was able to assure her that it would return in the course of about six months.

Mr. DOWSETT also mentioned a similar case, one which occurred in the lower premolar region under circumstances very similar to the case which Mr. Hopson had just mentioned. The patient was a man aged about 60. He was edentulous in the lower jaw, and apparently from no reason whatever—he had never worn a denture—he suddenly developed pain and swelling in the right premolar region. After this had gone on for several months he attended Guy's Hospital. When he saw the patient he learned that the swelling had only just burst, leaving a sinus. On probing down the sinus one could evidently feel enamel. He operated upon the man, and removed quite easily the right premolar. The lower premolar was perfectly normal except for a slight exostosis at the end. He thought the case had some bearing on the question of pain. It seemed to him that the pain and trouble were due to inflammatory mischief around the end of the tooth, starting probably from the exostosis. He also remembered another case which came under his attention some seven or eight years ago, in which the tooth was in the second lower molar region. When removed it had a very similar appearance to that which had been thrown on the screen. On cutting a section it was found there was an abnormal growth of the tissues—no ingrowth of the bone, but an abnormal growth of the enamel on the root portion mixed up somewhat with the cementum. With regard to whether or not Dr. Baker's specimen could be called an odontome, it seemed to him the time had arrived when there should be a very strict classification of odontomes. Personally he saw no reason whatever why any abnormality in the dental tissues should not be called an odontome, and also, wherever it was found that the enamel or dentine or cementum were out of place that should be called an odontome.

Mr. BALDWIN remarked that in such cases, whenever there was a channel leading from the buried tooth to the surface, it was likely that the irritation was due to germs in the potential space between the enamel and the soft tissues. That led him to remark that he hardly thought it was correct to say that the exostosis was a probable cause of irritation. Surely the exostosis was more a result of the irritation and the inflammation generally set up by germs getting in from the saliva.

Dr. ARTHUR BAKER, in reply, said with regard to the President's observations, when he suggested the specimen was an odontome, he did so in a very modified sense. He by no means meant to imply that the condition was a form of tumour which Mr. Bland-Sutton had defined so extremely well. He thought the condition he had presented suggested the appearance of what Mr. Salter had called a warty tooth. With regard to Mr. Hopson's remarks he was extremely glad to hear from him that he had had many similar experiences to his own. On several occasions, both in private and in hospital practice, he himself had had a good many cases in which it was very difficult to see why buried teeth should give rise to so much pain. Mr. Dowsett mentioned a case in which a bicuspid had given a considerable amount of trouble. He himself remembered a case of an old gentleman of over 70 years of age, for whom he had had to remove a buried bicuspid which was causing a great deal of pain and a considerable abscess around the tooth.

A Classification of Dento-facial Irregularities.

By J. SIM WALLACE, D.Sc., M.D., L.D.S.

THE classification of irregularities of the teeth is rather a thorny business, and I hope the classification which I am about to suggest may stimulate discussion—for whether good or bad in its present form, I am sure a little discussion may be likely to lead to a classification which will be more generally useful than any classification which has hitherto been made. There is not much originality in what I have to suggest, but rather an attempt to coördinate the best feature of the various classifications which I have come across. Possibly I may offend the partisans of each of those classifications, but, if so, it is certainly not my intention to do so.

A classification of dento-facial abnormalities should be based on facts and not upon erroneous assumptions. It should, as far as possible, be simple and natural or descriptive. It should, if possible, follow some method, and finally it should be of value from the point of view of diagnosis, etiology and treatment. There have been many classifications suggested, but few have met with general approval. That suggested by Dr. Angle has, however, attracted some attention; but this, too, must be discarded, not so much because it is highly artificial and difficult to memorize, but because it is based on fundamentally erroneous assumptions, and finally it is very incomplete. We now know that the first upper molar is by no means a fixed point, and that, moreover, the limitation of the classification to the occlusion of the teeth and not having a corresponding regard for the development and normality of the bodies of the jaws, render it quite impossible from a practical and scientific point of view. At one time I believed that no classification could be admissible if it were not purely etiological, but this is no reason for ignoring the fact that a classification may also be of great value from the point of view of diagnosis and treatment. It therefore has occurred to me that a classification primarily depending upon diagnosis and treatment, and secondarily on etiological considerations, might be the most desirable, since by this method we can most readily determine exactly what we have to deal with and see how we may change the results of abnormal processes and restore, as far as may be, the normal. It is, moreover, extremely simple, and recognizes the general truths contained in the classification of irregularities which has gradually been more or less consciously evolved during many years of progress in dental science.

This classification suggested itself to me while reading a valuable communication by Dr. Gires, of Paris, who contended that it was necessary to appreciate thoroughly both the classifications of Dr. Angle and Dr. Case. But while I appreciated Dr. Gires' conclusion that these two classifications are each the complement of the other, and that it is necessary to have studied each thoroughly, yet I felt that it was getting a little too cumbersome and, moreover, that both classifications contained a great deal that might be eliminated with advantage.

That the face should have as much consideration as the occlusion will be recognized by all, and therefore it should be noted both in profile and in full face, just as should the occlusion in front and at the sides be noted. As a matter of routine the face should be observed firstly from the front, secondly from the side, and then, thirdly, the models and occlusion should be considered in detail. This routine suggests the following groups:—

GROUP 1.—*Full face:*

Open bite.
Over bite.
Contracted arch.

GROUP 2.—*Profile:*

Superior protrusion.
Superior retrusion.
Inferior protrusion.
Inferior retrusion.

(In this classification the four divisions are given in ideal separation; in practice two of the divisions are frequently combined.)

Each division of the profile group is subject to three subdivisions. viz.: Total, Partial, and Unilateral.

(1) *Total* when the whole mandible or maxillæ are involved, with corresponding antero-posterior malocclusion.[1]

(2) *Partial* when, from insufficient or arrested development of the mandible or maxillæ, the teeth are deflected from their normal axes or retained in positions of arrested development, *e.g.*, flaring or protrusion of incisors, backward deflection or retrusion of incisors, outstanding canines, displaced premolars and impacted wisdom teeth.

(3) *Unilateral* when the irregularity is on one side only.

Now although this completes the classification of dento-facial irregularities, we have also, of course, *irregularities of individual teeth* with the dental arch normal. In this case there is no facial abnormality. I mention this group last and altogether distinct from the other groups in order to emphasize the fact that irregularities of individual teeth are not related to dento-facial irregularities either etiologically or from the point of view of prevention and treatment.

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[1] Here I should perhaps mention that what Dr. Angle calls mesiodistal malocclusion ought to be called antero-posterior, as the occlusion which he means by mesial and distal is neither mesial nor distal, but anterior and posterior.

In any classification which makes a claim to be of practical value it is necessary to be able to give the exact relation of the teeth to each other and to the body of the mandible or maxilla in which they are placed. For my present purpose it is sufficient to indicate how the position of the first molar may be recognized, this tooth having been admitted to be the most important in the dental arch since the classical works of Dr. Bogue, of New York, and Dr. Davenport, of Paris, were published. I need make no apology for dwelling a little on the exact position of the first molar, for all will agree with me when I say that anyone who treats a case of irregularity of the teeth without recognizing its position in relation to the body of the bone in which it is placed, and in relation to its fellow above or below, ought to be considered as "guilty of infamous conduct in a professional sense."

To determine the position of the first molars the following methods may be pursued. If the subject is young all that is required to be done is to examine the teeth of the temporary dentition and observe the amount of crowding. This may be calculated from the amount that the spaces which normally should exist in the temporary dentition have been obliterated by the crowding forward of the teeth from behind, and generally to a certain small extent also by the lack of intermaxillary development. In many cases approximal caries or extractions have to be considered, for it often happens that approximal caries allows the first permanent molar to be pushed forward, and this may occur in both the upper and lower arch. The amount of contraction thus observed must, of course, be calculated in addition to the amount obliterated by the crowding out of existence of the normal spaces between the temporary teeth. The models of both upper and lower teeth should, of course, be studied in arriving at a diagnosis from this point of view.

When there has been extraction of temporary teeth it must be observed whether the space remaining is equal to the normal mesiodistal diameter of the missing tooth plus the normal spaces between the neighbouring teeth. Similarly, when most of the permanent teeth have erupted, the amount of anterior displacement of the first molar may be most certainly gauged by the lack of space for the normal arrangement of the teeth. If, for example, a canine or premolar is completely out of the arch formed by the rest of the teeth it may be presumed that the position of the first molar is the breadth of a whole tooth forward in relation to the body of the mandible or maxilla, unless it is observed, from the direction of the incisors, that there has been retrusion of the crowns of these teeth. This does occur more or less at times, and must be considered if accurate diagnosis of the malposition of the first permanent molar is to be made.

Another method which might be employed is an application of Hawley's tables, but this I cannot go into just now. If it is found that the molar teeth (being in normal occlusion) are, say, the breadth of a tooth too far forward, if by expansion and general treatment of the case there is no reasonable prospect of pushing it backwards at least the breadth of half a premolar tooth, then the treatment will be not only mechanical but surgical also. In other words we have in this a rule for the extraction of four premolars, an operation which will continue to be justifiable, notwithstanding all that is said to the contrary, until some method is devised to stimulate the growth of the mandible or maxillæ sufficiently to make up for its arrested development.

DISCUSSION.

Mr. G. NORTHCROFT thought Dr. Wallace's choice of words in the subdivisions of his profile group was a little unfortunate. "Partial" and "unilateral" seemed to him to be confusing terms, as Class 3 might clearly include Class 2. It seemed to him also that the subdivision 1 might obviously be done away with, as a given condition must be either total or partial.

Dr. BAKER thought the paper had to be read several times before the points contained in it could be assimilated. He was very much interested to hear that Dr. Wallace had not given up the idea of extracting. He thought that was a thing which was apt to be ignored in these days, when people were so full of treating irregularities and retaining all the teeth. He thought the possibility of really being able to jump a bite was extremely doubtful. He had tried it on a great many occasions, and he had to confess he had had more failures than successes.

Mr. SCHELLING inquired under what classification Dr. Wallace would put an edge-to-edge bite.

Mr. D. P. GABELL asked Dr. Wallace what his normal standard was from which he measured, and what measurements he made.

Mr. RUSHTON inquired under what classification Dr. Wallace would put those cases in which there seemed to be undue prominence of both jaws; and also, in deciding as to any standard of beauty, whether he adopted the Grecian, the Hottentot, or any other style. Each had their own standard, and he should like to know what the standard of Dr. Wallace was.

Mr. BALDWIN thought in regard to the last point it was not necessary to go into such details, but just to judge by one's sense of symmetry and one's sense of what should be the normal positions and relations of the teeth to the rest of the face, and particularly the profile.

Mr. J. F. COLYER thought it was extremely difficult to get a classification which embraced all conditions. He did not agree with Angle's classification, because it did not take into consideration the question as to whether one was dealing with maxillary bones which had been arrested or interfered with in their development. It would be of great advantage, he thought, to obtain a classification which was etiological, but he failed to see how that could be arrived at. It seemed to him, therefore, that the most practical way to deal with the subject was to have a classification which was partly etiological and partly clinical. He thought irregularities of the teeth fell into three well-defined groups. First of all there was the group associated with perfectly or normally developed bones, and in that group, he thought, went those various forms of irregularities of individual teeth. Into that group also fell a very important type of irregularity which, he might say, Angle did not refer to—those very difficult cases of unerupted canines and misplaced third molars. There was the second group of irregularities, associated with some interference in, and lack of, the development of the bones. We could not, with our present knowledge, subdivide this group on etiological lines, and therefore it would be wise to subdivide it on clinical lines. It seemed to him, clinically, cases of irregularity associated with some arrest in development of the maxilla fell under five headings. First of all there was the general crowding, and secondly and thirdly there were the superior protrusion and inferior protrusion. Then there were open bite and the ordinary cross bite. But there was another very important class of irregularity which must find a place in any classification, namely, the class of irregularity which was associated with malformations—congenital malformations of the jaw—such as arrested development of the mandible. It did seem to him that if there was to be a classification which embraced all irregularities met with, the simplest way would be to divide it first of all into an etiological classification and then subdivide under clinical varieties. He assumed that the use of classification was to be some guide in treatment. As far as he could see, in Angle's classification there was no attempt made to deal with the question of the defective development of the jaws and also no attempt made to deal with those difficult cases where there was definite interference in the maxilla conditions. In Angle's work one looked in vain for description and treatment of really difficult cases.

Dr. SIM WALLACE, in reply, said he was inclined to agree with Mr. Northcroft's criticisms that "total" was unnecessary. It was obvious that if a thing were total it was hardly worth mentioning that it was so. On the other hand, when making the classification, he had thought it necessary.

Mr. Northcroft also said that the use of the terms "partial" and "unilateral" was unfortunate, and perhaps it was. He would like someone to suggest two better words. With regard to Mr. Schelling's question in reference to edge-to-edge bite, it was, he thought, quite evident where that was included in the classification; it was, as a rule, simply a slight inferior protrusion; sometimes, however, it might possibly be that the upper teeth were slightly in retrusion. With regard to Mr. Gabell's question as to what was the normal standard, he would like to leave that to the operator's good taste, to his knowledge of the anatomy of a well-developed mandible and maxillæ, and to his power of recognizing the normal relation of the jaws to the teeth and of the occluding teeth to each other. With regard to undue prominence of both jaws, it was, as a rule, apparent rather than real, and was due to a combination of superior and inferior protrusion, which were usually "partial"—the jaws being rather undeveloped and the anterior teeth deflected forward. When it was "total," both jaws and dental arches were abnormally far forward, at least according to our European standards.

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PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE

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CONTENTS.

December 7, 1907.

PAGE

Presidential Address. By PETER MCBRIDE, M.D. 1

Cases and Specimens:—

Extirpation of the Labyrinth. By SYDNEY SCOTT, M.S. 5

Foreign Body in the Ear. By MACLEOD YEARSLEY, F.R.C.S. 6

Effect of Treatment in a Case of Sudden Deafness occurring whilst the Patient was under Treatment for Tertiary Syphilis. By RICHARD LAKE, F.R.C.S. 7

Nine Specimens of Fracture through the Temporal Bone. By ARTHUR CHEATLE, F.R.C.S. 10

A Deaf Mute, aged 11, apparently regaining her hearing. By L. A. LAWRENCE, F.R.C.S. 13

Stenosis of Right Auditory Meatus. By W. H. KELSON, M.D. 14

Two Cases of Epithelioma of the Ear. By HUNTER TOD, F.R.C.S. ... 15

Demonstration of Specimens. By WILLIAM MILLIGAN, M.D. 18

February 8, 1908.

Cases and Specimens:—

Histological Preparations of the Human Cochlea. By SYDNEY SCOTT, M.S. 19

Keloid following Mastoid Operation. By HERBERT TILLEY, F.R.C.S. ... 20

Unusual Case of Cerebellar Abscess. By D. R. PATERSON, M.D. ... 23

Cases and Specimens (continued) :—

PAGE
Right Temporal Bone showing how Pus may reach Neck without passing through the Mastoid Process. By A. H. Cheatle, F.R.C.S. 24
Ménière's Disease in a Girl aged 14. By R. Lake, F.R.C.S. 24
Case for Diagnosis. By R. Sturgeon Cocke, F.R.C.S.Ed. 27
By Hunter Tod, F.R.C.S. :—
(1) Vertigo, the Symptoms pointing to some Inflammatory Lesion within the Semicircular Canals 28
(2) Lateral Sinus Thrombosis; subsequent Meningitis; Recovery ... 30
Pulsating Growth in Left External Auditory Meatus. By J. Barry Ball, M.D. 32
Primary Epithelioma (?) of Tympanum following Chronic Suppurative Otitis media. By A. L. Whitehead, B.S. 34

March 7, 1908.

The Operative Surgery of Labyrinthitis, based upon an experience of thirty cases. By C. Ernest West, F.R.C.S., and Sydney Scott, M.S. 37
Deafness resulting from Epidemic Cerebrospinal Meningitis. By H. H. B. Cunningham, F.R.C.S.I. 83

Cases and Specimens :—

Purulent Otitis media with Deep Ulceration of External Meatus in a Case of Secondary Syphilis. By Adolph Bronner, M.D. 88
Crossed Abducens Paralysis in a Case of Cerebellar Abscess. By D. R. Paterson, M.D. 88
Branchial Sinus leading into External Auditory Meatus; Sinus Excised. By C. H. Fagge, F.R.C.S. 90
Epithelioma of Middle Ear (? Traumatic). By W. Milligan, M.D. ... 91
Suppuration in the Right Labyrinth; Operation; Recovery. By Macleod Yearsley, F.R.C.S. 92
Specimens and Photographs of Pathological Conditions found in the Labyrinth. By Albert Gray, M.D. 93
Histological Preparations of the Human Labyrinth. By Sydney Scott, M.S. 94
Specimens of the Temporal Bone illustrating Operations on the Labyrinth. By C. E. West, F.R.C.S. 94

May 2, 1908.

The Clinical Pathology of Aural Discharges. By Wyatt Wingrave, M.D. ... 95

Cases and Specimens (continued):—

PAGE

By W. S. Syme, M.D.:—

(1) Lateral Sinus Disease; Operation; Cure 123

(2) Bezold's Mastoid Empyema which Discharged into the Pharynx ... 125

(3) Injury to the Pharyngeal Portion of the Eustachian Tube from Operative Procedures 126

By J. Dundas Grant, M.D.:—

(1) Unilateral Hysterical Nerve Deafness of Sudden Onset, with Hemianæsthesia and other allied Stigmata 127

(2) Unilateral Nerve Deafness in an Elderly Man 130

Specimens of Necrosis of the Labyrinth. By P. Macleod Yearsley, F.R.C.S. 131

Chronic Eczema of both Auricles and Meatuses with unusual degree of Hyperplasia in a Woman aged 43. By E. Furniss Potter, M.D. ... 132

Complete Occlusion of the Posterior Nares with flattening of the Nose externally and almost complete Absence of the Nasal Septum. By Hunter Tod, F.R.C.S. 133

June 27, 1908.

Cases and Specimens:—

Middle Ear Suppuration; Phlebitis of Lateral Sinus; Cerebellar Abscess. By P. McBride, M.D. 135

By A. Logan Turner, M.D:—

(1) Patient operated upon for Sigmoid Sinus Thrombosis 137

(2) Patient after operation for Left Temporo-sphenoidal Abscess occurring in the course of a recent Middle Ear Suppuration ... 137

(3) Two Patients after the Mastoid Operation, with preservation of the Tympanic Membrane and Ossicles 144

Preparations from a Case of Right Temporo-sphenoidal Abscess and Left Sigmoid Sinus Thrombosis. By A. Logan Turner, M.D., and Henry Wade, F.R.C.S.Ed. 138

A Case of Infection of the Lateral Sinus; Pneumonia and Pericarditis; Ligature of the Jugular Vein; complete Recovery. By W. Permewan, M.D. 139

An Unusual Sequel to the Radical Mastoid Operation. By W. G. Porter, F.R.C.S.Ed. 144

Notes on Partial Grafting with the aid of Local Anæsthesia in the After-treatment of the Radical Mastoid Operation. By J. Stoddart Barr, M.B. 145

A Patient after Vestibulotomy (Right Ear). By J. S. Fraser, M.B. ... 149

Notes of a Case of Ablation of both Vestibules for the relief of Vertigo. By George Gibson, M.D., and Richard Lake, F.R.C.S. 150

Cases and Specimens (continued) :—

PAGE
Notes of a Case of Otitic Meningitis with Histological Specimens of the Labyrinth, demonstrating Streptococci in situ. By SYDNEY SCOTT, M.S. 157
Preliminary Note on Cytological Examination of the Discharge in Cases of Middle Ear and Maxillary Sinus Suppuration. By JOHN M. DARLING, M.B. 163

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Otological Section.

December 7, 1907.

Dr. Peter McBride, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

Gentlemen,—I should like in the first place to express my deep sense of the honour you have done me in electing me President of this Section. I can assure you it was as gratifying to me as it was unexpected, and I can only add that during my tenure of office I shall do my best to prove worthy of the confidence you have been kind enough to repose in me. To-day, as we inaugurate the meetings of the Otological Section of the Royal Society of Medicine, we must not forget how much we owe those Societies which were not only its predecessors, but one might almost say its progenitors. I refer, of course, to the Otological Society of the United Kingdom and to the British Laryngological, Rhinological and Otological Association. Such societies as these, existing as they do in all civilised countries, have had much to do with the marvellous strides which scientific otology has made of late years.

There is no stimulant for thought equal to free discussion, nothing sharpens the logical faculties more, and there are few better incentives towards wide reading. Most of us here have been engaged in the practical work of otology for some years—a few of us have been so engaged for several decades. When I look back over a period of nearly thirty years and remember the modes and methods of those times and compare them with those of to-day, the question sometimes shapes itself: "What of the next thirty years?" He would indeed be a rash man who would venture to forecast the paths along which progress will be made, but we may feel tolerably sure that as advances have occurred in the past, so they will take place in the future.

It seems to me that we shall not be far wrong if we assume that progress has followed a great general law. It has resulted from the application to otology of general medical and surgical principles and methods, modified, of course, to suit the peculiarities of the region to be dealt with, but always resting, so far as circumstances have permitted, on a sound basis of anatomy, physiology and pathology—a trilogy which gives the most certain guarantee for rational therapeutics. Having gone so far, it may be interesting to speculate upon the further lines of advance.

Reasoning from the past, we shall feel rather doubtful whether we can expect any great advance in anatomical knowledge, and we may feel tolerably certain that such advance as occurs will be in microscopic rather than naked-eye anatomy. In physiology, on the other hand, there still remains something to be done, and we shall all look forward to further light some day to be thrown on the cochlea and semicircular canals, the functions of the ossicles, and other matters still in dispute. While every intelligent aurist keeps himself abreast of modern anatomical and physiological work, yet his interest tends to centre more in morbid anatomy, including under this term bacteriology and cytology. We all know what valuable additions have been made to our knowledge both in non-suppurative and suppurative forms of ear disease.

In dealing with chronic middle ear deafness the older aurists—and I include some distinguished authors whose works were widely read even within my own recollection—tended to evolve pictures of disease founded rather upon their own subjective impressions than upon well-ascertained facts. Clinical observation was largely employed in arriving at morbid anatomy. Now, however, all this is changed. Gradually a good deal of laborious work has resulted in records of a certain number of cases accurately observed during life and minutely examined after death. Thus we are approaching a knowledge of chronic deafness due to non-suppurative conditions more in accordance with scientific fact.

In suppurative ear disease we have long known the general pathology of the condition, but, as we are all aware, careful microscopic work combined with bacteriology and cytology has thrown much new light upon both the ear disease and its complications.

If we turn to the older works we shall find that to a great extent what the authors lacked in knowledge of pathology, and even pathogenesis, they atoned for by accurate clinical observation. Yet even here we have made great advances. Merely to give a few instances, I may call attention to the modern methods of testing hearing with tones of different pitch. The seniors among us will remember how delightfully dogmatic we used to be over the tuning-fork test. Certainly we occasionally received an awkward shock when we found Weber's experiment in conflict with Rinné's, but, like theologians confronted with awkward problems, we still kept our faith in one or other, if not both. Now, however, we should never dream of expressing an opinion founded solely on such tests, but make a point in all doubtful cases of investigating the upper tone limit and the ability to hear sounds of low pitch. It will be remembered that quite a considerable number of cases in which the organ of Corti was examined after death have been recorded, and that the results go to show that in middle-ear deafness the low notes are lost, while in affections of the cochlea the upper tone limit is lowered. Again, in cases in which disease of the vestibular apparatus is suspected, we now carefully examine the static sense. It is also in many instances important to watch for the occurrence of nystagmus, and it is even asserted by some that in modifications of this symptom we may have a valuable method of differentiation between cerebellar and labyrinthine disease. While, therefore, there has been much careful observation of symptoms both by older and by new workers, we may confidently expect that in the no distant future much will be added to our knowledge of semeiology and diagnostic methods. To us, as practical aurists, however, anatomy, physiology, pathology, and even clinical observation are merely means to an end—the cure of ear disease. Speaking broadly, we may say that of late years the striking advances in treatment have been made in suppurative disease, while infinitely less success has attended endeavours to relieve the effects of chronic non-suppurative middle ear affections.

We all know how, from simply opening the antrum, we have passed through various stages until we have arrived at the radical mastoid operation of to-day. Neither need I trace the gradual evolution of surgical methods which enable us now to treat thrombosis of the lateral sinus, intracranial abscess, and even meningitis. The immense progress we have made in the treatment of middle ear suppuration is an excellent illustration of the suggestion I ventured to make that progress in our specialty—as, indeed, in all others—depends upon applying to the part general medical and surgical principles.

I have said that the most important therapeutic advances have been made in the treatment of suppurative disease, and this is the case with one exception. I refer to the recognition of naso-pharyngeal adenoids as a cause of chronic deafness. While the radical operation and its further developments have saved many lives, it is safe to assert that the adenoid operation has saved infinitely more ears. Here again we are acting on well-understood principles, and good results follow. As you are aware, a considerable number of operations have been from time to time recommended which are, if I may say so, more specialised in character. Thus it has been proposed to modify the radical operation by leaving the membrane and ossicles; further, ossiculectomy has been advocated both for suppurative and non-suppurative conditions. I should unduly extend these introductory remarks and, moreover, I should be guilty of a breach of manners were I from this chair to enter upon controversial questions. I think, however, that even those gentlemen who advocate the methods I have referred to will admit that their application must, from the nature of things, be very limited. Thus, opening the antrum and leaving the membrane and ossicles presupposes that the tympanic attic and incus are free from disease. Again, removal of the ossicles alone can only be curative in those cases in which, after the operation, attic and antrum drain freely. While in non-suppurative conditions improvement in hearing may follow the removal of the malleus and incus, if the stapes be mobile—as sometimes occurs after healed suppuration, and occasionally in adhesive processes due to catarrh—it can have no beneficial effect in otosclerosis where the lesion lies in fixation of the stapes and osteo-porosis of the labyrinthine capsule.

Other methods of treatment, midway between the operative and non-operative—such as suction with the double object of drainage and congestion, the application of Bier's congestion method to the ear, but from the nature of things also to the brain, and electrolysis of the Eustachian tube—are some of them still on trial, although, speaking for myself, I cannot think they will have a great future.

As we get further away from surgery, we find that progress is less marked. In the employment of drugs we have not made great advances. The application of local anæsthetics and the introduction of pilocarpin for certain cases of labyrinthine deafness seem to me to be of value, but, speaking generally, we are where we were years ago, with this difference, that most of us are more sceptical about drugs, and therefore less inclined to use them without very definite indications.

I have thus sketched in cursory form, and as briefly as possible, some of the salient developments of modern otology in the past, and I must admit that it is difficult to foresee room for advances of anything like such a striking character in the future. It almost looks as if we shall be thrown back upon developing knowledge of details anatomical and physiological, but above all pathological and clinical. It does not appear to me that we can hope for great new therapeutic triumphs, because analogy leads us to expect them only from an extension of surgery, and it would seem that we have come almost to the possible limits in this direction. History, as a whole, however, contradicts this pessimistic view.

and although at present we cannot exactly forecast the amount and direction of the new light which will fall upon our specialty, we may rest assured that sooner or later it will come, and I feel sure that in its coming it will be materially assisted by the workers in this Section.

Case of Extirpation of the Labyrinth.

By SYDNEY SCOTT, M.S.

B. T., a wardmaid, aged 20. Bilateral otorrhœa from infancy. Very deaf for three years. First seen June, 1906. In November, 1906, complete tympano-mastoid operation on the left side. January, 1907, complained of the right side. Giddiness and fainting fits. February: First fell down, apparently unconscious. March: Complete tympano-mastoid operation, right side. No fistula of the external semicircular canal. Vestibular window and fossula rotunda not explored. After operation tympanic granulations persisted. June: Readmitted with headache, giddiness, vomiting, rigor, pyrexia, furred tongue, anæmia, and loss of flesh. Leucocytosis, 14,000. Mastoid cavity reopened. No extra-dural abscess. Lateral sinus and brain natural. Major symptoms subsided, but vertigo evident during convalescence in July. Coördination tests—Subjective sensation: Objects moving from the right to the left, horizontally when head erect; diffuse giddiness when lying down. Static: Swaying of the body irregularly with eyes closed when standing unaided. Dynamic: Attempting to walk with eyes open sways towards the right without falling. With eyes closed falls to the right unless supported. Jumping tests cannot be carried out owing to instability. Hearing tests: Absolute deafness with complete perosseous loss on the right side. Deaf to conversation and the watch on the left side. Weber to the left, Rinné negative on the left. August: Operation on right labyrinth. Tympanum reopened, whole of osseous meatal wall removed, and parotid gland drawn forwards. External arcuate eminence normal. Fenestra ovalis explored with fine probe, and footplate felt. Fossula rotunda explored; probe entered by its own weight into the vestibule. Cochlea and vestibule freely opened with gouge, and found to be represented by granulation tissue. (Histological preparation of contents of cochlea shown.) Cerebro-spinal fluid escaped for twenty-four hours. Slight temporary facial weakness followed. Healing of cavity uninterrupted. Giddiness completely disappeared.

DISCUSSION.

Dr. W. Milligan congratulated Mr. Scott on the excellent results which he had obtained in his cases. He thought one of the lines of progress in the specialty would be the surgery of the labyrinth. He believed a considerable number of cases of labyrinthine suppuration were passed over in practice, and the Section should welcome such an excellent communication as Mr. Scott had now brought forward. He desired to emphasise one phrase in the paper, "osseous meatus removed." He regarded that as a most valuable part of the operation, as it admitted of a better view of the deep portion of the labyrinth. He asked whether Mr. Scott was in the habit of allowing the large cavity left as the result of the operation to fill up entirely with granulation tissue, or whether he had ever attempted to graft the cavity. He also asked whether Mr. Scott operated on such cases by means of a fine burr or chisel.

Mr. Scott, in reply, thanked Dr. Milligan for his remarks. In his cases the cavities were allowed to granulate and cicatrise without skin-grafting. Both were operated upon with the gouge, not with the burr. In all the cases on which he had operated he used the long-handled straight gouge, with a diameter of $\frac{1}{2}$ cm. to $\frac{1}{3}$ cm. He thought that if the floor and the anterior wall were removed and the cochlea were destroyed, as there was no hope for the patient's hearing there was really no advantage in causing the cavity to become lined with epithelium. It seemed better to allow the cavity to become contracted, as it did after removal of the osseous meatus, sometimes filling up with scar tissue flush into the concha.

Microscopic Specimen and Drawing of a Foreign Body.

By Macleod Yearsley, F.R.C.S.

S. B., aged 42, came to hospital on September 4, 1907, giving the following history: One week previously he was lying on the grass in Devonshire when, in rolling over, a head of grass went into his right ear and broke off, causing him great pain. He plucked the grass from his ear, but had experienced much pain and discomfort in the ear since.

On examination the meatus was wide and straight. The right membrana tympani exhibited the following appearance: Anterior and posterior to the handle of the malleus were large white plaques. In the anterior inferior segment was a small, round, dry perforation, with a pinkish edge. Immediately behind this perforation was a small brownish projection, not unlike a small blood-clot, which was hard to the probe. Gas was administered and the foreign body was separated by means of the myringotome and removed with forceps. The ear was packed for twenty-four hours, and the patient recovered without any complication.

The body, which is shown under the microscope, proved to be a small grass seed, the fine curled rootlet of which was growing through the membrane into the middle ear. When first removed the circle formed by this rootlet was intact, but it was unfortunately broken in the mounting.

DISCUSSION.

Mr. C. E. WEST asked what was meant by the words "in the membrana tympani." Did it mean that it was embedded in it, and if so, to what depth? Or was the seed in a perforation, or on the membrana tympani?

Mr. W. H. BOWEN, in reply, said he saw the patient with Mr. Yearsley when he first came. The seed was embedded in the membrana tympani and was thought to go through to the other side. The perforation was caused by the seed.

Notes on the Effect of Treatment in a Case of Sudden Deafness occurring whilst the Patient was under Treatment for Tertiary Syphilis.

By RICHARD LAKE, F.R.C.S.

A. C., aged 27, potman. Seven months before coming under observation the patient contracted syphilis, and at once attended at the Lock Hospital. Four months after he had been under treatment he began to get slight attacks of vertigo with a tendency to fall to the left side. One month later, *i.e.*, when he had been under treatment five months, he noticed he was getting deaf. In four days the deafness was complete, and there was also tinnitus of a hissing variety, both ears being affected. Two months later, after seven months anti-specific treatment, he presented himself for treatment at the Royal Ear Hospital.

October 17, 1907: The patient, who was very anæmic, complained of deafness and tinnitus of two months standing, both ears being affected. There was no pain or discharge. On examination both membranes were slightly indrawn and the cone of light was broken on both sides. There was no other pathological condition to be observed. Tests for hearing:—

		Right	Left
Acoumeter		½ in.	Contact
Voice		A very loud voice, only heard on contact	A very loud voice, only heard on contact
Whisper		0	0
Rinné C.		Positive	Positive
C. mastoid		—30 sec.	—30 sec.
3C 16		0	0
2C 32		0	0
1C 64		Perception	Perception
C 128	Air conduction	—40 sec.	—55 sec.
C^1 256			
C^2 512			
C^3 1024		Very diminished	Very diminished
C^4 2048			

Treatment.—Mercury and iodides were stopped at once, and the following treatment prescribed: Pil. iodoform. gr. iij., three times a day, together with an iron mixture.

Progress.—The patient attended the hospital regularly once a week, and from the first showed signs of improvement, and on November 28 presented himself at the hospital, saying he was quite well. There were one or two slight attacks of vertigo during the time he was under treatment.

November 28: Patient is looking much better and has almost lost the anæmia which was such a marked feature of the case at his first attendance. Tests for hearing:—

		Right	Left
Acoumeter ...		$8\frac{1}{2}$ in. ...	1 ft.
Voice ...		Over 16 ft.	Over 16 ft.
Whisper ...		3 ft. ...	2 ft. 4 in.
Rinné C. ...		Positive ...	Positive
C. mastoid ...		—6 sec. ...	—10 sec.
3C 16	Air conduction	Slightly diminished ...	Slightly diminished
2C 32			
1C 64			
C 128			
$C^1$256		Normal	Normal
$C^2$512			
$C^3$1024			
$C^4$2048			

The case was brought forward as one of apparently severe internal ear disease, the result of syphilis which had, in a large measure, recovered as a result of treatment, although it was a matter of consideration whether the condition was specific, toxic, or the result of anæmia, or due to all three combined.

DISCUSSION.

Mr. CRESSWELL BABER asked whether inflation was practised in the case in order to exclude middle ear disease. He understood that bone conduction was minus 30, and air conduction minus 40; in one ear, therefore, it seemed that besides the nerve deafness there might be some middle ear trouble.

Dr. DUNDAS GRANT asked what was the duration of the deafness, and how long after the primary syphilis it came on, also whether the diagnosis of primary syphilis was quite certain. It seemed very early for tertiary syphilis.

Dr. MILLIGAN said that from the notes read he regarded it as a somewhat late case of secondary syphilis of the auditory nerve in a debilitated subject, the kind of case in which pilocarpin produced good results. He asked whether it had been used.

Dr. A. BRONNER said the case opened up a very important subject, namely, whether syphilis often affected the hearing. Common sense would lead one to think it did, but according to experience and the statements in the text-books it did not often do so. It would be a good thing some day to have a discussion on the subject. Cases were constantly occurring in which a man had had syphilis and then he got deaf: and the question arose whether the deafness was due to the syphilis.

Dr. ALBERT GRAY did not think there was much doubt that syphilis caused deafness. Everyone knew the hereditary cases which came on with interstitial keratitis. He remembered a case in a man who undoubtedly had acquired syphilis, and was suffering at the time from an ulcer of the leg which only iodide of potassium healed. He had giddiness, deafness and staggering, and that was the only case in which he had seen labyrinthine deafness of syphilitic nature cured. The deafness following syphilis appeared to be an affection of the auditory nerve, not the labyrinth.

Mr. E. B. WAGGETT said he could mention a case of the same kind as Mr. Lake's, in which one ear practically recovered but the other remained deaf. There had been very marked vertigo, throwing the man on to the ground. The deafness of one ear and the vertigo were cured by antisyphilitic treatment in the course of a few weeks.

Mr. A. CHEATLE said cases of deafness due to early syphilis were not uncommon, and he had seen a fair number of cases. It was usually associated with giddiness, vertigo, and profound deafness. It was a pity to class the lesions into secondary and tertiary: he did not think that syphilis admitted of being put into such strict categories. He thought in these cases that there was an effusion into the labyrinth comparable to that in iritis. If they were seen early, prognosis was good.

Dr. URBAN PRITCHARD said that all things were comparative, and it must be admitted that cases of deafness caused by syphilis were comparatively rare. He agreed that there should not be rigid lines laid down dividing syphilitic lesions into secondary and tertiary, and in the cases where there was marked nerve deafness in the later stages much benefit sometimes followed the Aix treatment. He remembered a case in which there was perfect resolution on one side, but not the least effect on the other. Of course, it was most important for the patient to get hearing on one side. On that side it became perfect, although there was a little hyperæsthesia of the nerve afterwards.

Mr. A. L. WHITEHEAD said it was interesting to get as many opinions as possible failing a special discussion on the subject. Most were agreed that deafness, associated with congenital syphilis, was not uncommon; and those cases of syphilitic affections of the hearing which had been seen were almost invariably not associated with secondary syphilitic symptoms. The gummatous stage very rarely indeed affected the ear, and he would like to know whether any members had seen a case, excluding those associated with congenital syphilis.

Mr. DAN MACKENZIE said that they had also to remember that tabes was a late manifestation of syphilis, and that, therefore, nerve deafness referable to that and similar profound nerve lesions must also be looked upon as due to syphilis.

Mr. SYDNEY SCOTT asked if any of those present had had the opportunity of histologically examining the auditory nerve or the cochlea or the vestibule in cases such as those under discussion, and if so, what changes had been demonstrated.

The PRESIDENT (Dr. McBride) said that syphilitic deafness was a very interesting subject, and he agreed that it would be well to have a discussion thereon. He was surprised at the view that syphilitic deafness was believed to be so rare. He thought he had seen a good deal of it. Excluding the hereditary specific deafness associated with keratitis in children, there were many cases associated more distinctly with tertiary than with secondary syphilis. He believed the accepted view, advanced by Gruber, was that there was a small-celled infiltration of the labyrinth. There might be various forms of syphilitic deafness. First there was sudden nerve deafness, which he divided into two forms: one in which the cochlea appeared to be alone affected and which was called by Roosa syphilitic cochlitis. Another form was that in which there was not only deafness and tinnitus but also giddiness, seeming to show that both the vestibules and the cochlea were affected. Yet another variety was well illustrated by the following case: A man with a syphilitic history suddenly had a cold bath and turned deaf, without giddiness and with bone conduction retained. He had not seen any such cases of recent years, and therefore had not been able to test them with tones of different pitch. But he had in all seen a fair number of instances. The only cases he remembered in which anything approaching a definite cure resulted were one or two which were submitted to pilocarpin treatment, and one which had Aix treatment.

Mr. LAKE, in reply, said he thought many of the remarks were not for him to deal with. The middle ear had not been inflated. Seven months elapsed from the primary infection to the patient's coming to hospital; deafness occurred five months after treatment was commenced, and that was five months after the syphilitic infection. He could not verify the latter. The patient had had a considerable quantity of mercury. Pilocarpin had not been used. His wish had been to know what variety of toxic infection those present might consider it to be.

Nine Specimens of Fracture through the Temporal Bone.

By ARTHUR CHEATLE, F.R.C.S.

IN 1, 2 and 3 a double line of fracture separates the roof of the Eustachian tube, the middle ear and meatus from the rest of the bone, with tearing of the membrane. In 4 and 5, the fracture is much the same as in 1, 2 and 3, but the outer wall of the antrum is also separated. In 5 the separated portion is broken. In 6 the same injury is present, but the fracture extends into the groove for the lateral sinus. In 7 the roof and posterior superior walls of the meatus and outer wall of the antrum and upper part of the squama are separated in one piece; the fracture extending into the groove for the lateral sinus. The membrane is torn through above and the incus dislocated. In 8 it is much the same as No. 7, but the mastoid process is also separated. The membrane is torn across above and the incus lost. The roof of the antrum is missing. In 9 the fracture separated the outer wall of the Eustachian tube, the entire meatus, the outer wall of the antrum and mastoid process from the rest of the bone. The carotid canal, the lateral sinus groove and the jugular fossa are broken through. The facial nerve is torn through as it leaves the shelter of the external semicircular canal. Various parts of the two large pieces are broken off. Besides the fracture there is a curious malformation below the oval window. A thin bar of bone separates the window from what looks like a second oval window below leading also to the vestibule. In all the bony labyrinth is intact. The outpouring of the cerebro-spinal fluid which occurs in these cases is most probably due to tearing through of the dura mater of the middle fossa. The facial nerve is only torn across in Specimen 9, although it is exposed where it winds round the anterior extremity of the superior canal and just before entering the middle ear in Specimens 1, 2, 3, 4, 5, 6 and 8. The lateral sinus groove is involved in Specimens 6, 7, 8 and 9. The carotid canal is broken through in Specimen 9. The extreme deafness resulting in these cases is not due to fracture through the labyrinth, but to concussion, perhaps hæmorrhage into the labyrinth or tearing through of the auditory nerves.

DISCUSSION.

The PRESIDENT said that though the specimens did not lend themselves to much discussion he was sure he was voicing the sentiments of everyone in thanking Mr. Cheatle for bringing forward such a valuable collection.

Mr. L. B. RAWLING said that in 1904, in the Hunterian Lectures, he dealt with the subject of fractures of the base of the skull, the lectures being based on over 100 cases seen at St. Bartholomew's Hospital, 40 per cent. of which involved the region of the petrous portion of the temporal bone. After working at the subject for some time he concluded that there were only two forms of petrous fracture which were in the least common, firstly that fracture which, as the result of a blow on the side of the head, passed inwards along the tegmen tympani on the roof, and along the Gasserian fissure on the floor of the middle ear, towards the region of the Eustachian tube, and from thence along the petro-sphenoidal suture to the central weak spot in the basis cranii, the sphenoidal sinus. The exact anatomical situation of the fracture was best illustrated by what could be seen on the anterior aspect of the posterior fragment, namely, the posterior two-thirds of the middle ear, the Eustachian tube and tensor tympani muscle. The question of facial nerve implication was of very great importance, and, in this class of fracture, the nerve was either just involved in the region of the geniculate ganglion or just escaped. Any facial paralysis was consequently of a temporary nature only, and complete recovery could usually be prognosticated. Amongst Mr. Cheatle's specimens there was no example of the second typical group of petrous fractures, a group best illustrated by an example. A man receives a blow on the left occipital region, the fracture passing across the left cerebellar fossa and striking the foramen magnum immediately posterior to the left condyle and continued, from the opposite side of the foramen, first to the jugular foramen and then across the right petrous bone, traversing that bone in such a manner as to cut across the line of the facial nerve in the region of the geniculate ganglion. In this group the facial nerve was completely cut across, with resultant immediate and permanent facial palsy, associated with a variable degree of deafness from labyrinthine involvement. He was surprised to see in a recent American journal that a surgeon had denied the assertions which he (Mr. Rawling) had made in this connection. Mr. Sydney Scott, who was acting as Surgical Registrar at St. Bartholomew's Hospital, had, however, confirmed all the points, and Mr. Cheatle's specimens now definitely determined the points which he (Mr. Rawling) had investigated and enunciated in every particular.

Mr. SYDNEY SCOTT said that at one of the meetings of the Otological Society Dr. Milligan reported a case of hæmorrhage from the external auditory meatus, associated with fracture of the base of the skull, in which the bleeding came from the lateral sinus. He (Mr. Scott) mentioned at that time that severe continued hæmorrhage from the ear was more commonly derived from the middle meningeal artery, and not from either the tympanic membrane or the Eustachian tube, or from the lateral sinus. Last year he saw a boy who was admitted to St. Bartholomew's Hospital with the story that he had been sliding down banisters in a large building and had fallen more than 40 ft. on to a stone pavement below. From his ear escaped brain tissue. The brain tissue was examined microscopically for proof. The boy got well. Through Mr. Rawling he had heard of four other cases of the kind, two of which had also recovered. He would like to hear whether Mr. Cheatle had found brain exuding from the external auditory canal after fracture in any of his cases.

Dr. MILLIGAN said that the reason he brought his specimens referred to by Mr. Scott before the Society was to elicit an opinion with regard to prognosis. It was remarkable how very rarely there was an effusion of blood into the cochlea in such cases. In the majority of severe fractures the auditory nerve was torn. At the porus acusticus there was extensive hæmorrhage into the nerve, and if the patient lived for a short time and then died, the post-mortem showed a rapid young-celled infiltration between the fibres of the nerve. That fact was most important, because some of the cases recovered, and it explained why the prognosis was so bad with regard to hearing. Concussion was an inexact term, and it was difficult to know exactly what it meant. Probably in most of the cases there was some hæmorrhage. On the previous day he saw a case which was referred to him by an insurance company in which the statement was definitely made that facial paralysis had existed after the accident. It was now nine weeks since the accident, and the facial paralysis had entirely cleared off. The patient was quite deaf. He accepted the statement that there had been facial paralysis which had now cleared off, and it corresponded with the remark which Mr. Rawling had made as to the usual recovery of facial paralysis in such cases.

Mr. CHEATLE, in reply, said the Society was fortunate in having Mr. Rawling present to discuss the matter, as his work was well known in connection with fractures at the base of the skull. He did not agree with Dr. Milligan that deafness due to concussion was always the result of a hæmorrhage into the labyrinth. If it was so there would be vertigo: but in many cases of nerve deafness resulting from fracture of the skull there was no vertigo. Concussion was in some cases a nerve lesion.

Case of a Deaf Mute, aged 11, who is apparently regaining her hearing.

By L. A. LAWRENCE, F.R.C.S.

WAS not a strong baby; measles at seven months; went to school when aged 3; not deaf, could speak quite plainly. At 7 years of age teacher of school wished tonsils and post-nasal growths removed. This was done under gas at a hospital as an out-patient. When the child returned home she was quite deaf and did not speak. Between the ages of 9 and 11 she commenced talking again in consequence of having been at a school for lip-reading. She now speaks with a marked deaf-mute intonation. For the last year it has been noticed that there is some return of hearing. She can hear a loud voice quite close to the ears; better in the left ear. Can hear all the tuning-forks from 3C to C_3, but does not hear C_4. Rinné's test positive for C_3, C_2, C_1, C, and 1C; negative for 3C. Weber's test to left ear. Much loss of bone conduction for C_2, C_3, and C_1. Nothing noticeable about drum membranes. Never had discharge from the ears.

DISCUSSION.

Mr. CHEATLE said the fact that profound deafness came on immediately after the tonsils and adenoids were removed seemed to him to show that the deafness was functional rather than organic, and that fact might account for the recovery now taking place.

Dr. DUNDAS GRANT said his impression was that it was juvenile hysteria, although the present gradual recovery certainly seemed to point to the contrary.

He had a case in which absolute deafness took place, and the patient was so absolutely deaf for several years that she acquired the faculty of lip-reading. He was unable to cure her, but she suddenly recovered completely as the result of having to lie in bed for an intercurrent illness. The question was whether the present patient should be kept at a school for lip-reading. It would be a great pity if she were not placed among normal speaking children, so that by imitation she might acquire the natural mode of speech.

Mr. LAWRENCE, in reply, said that all he could judge from as to the previous condition of the hearing was from what he had been told. The deafness was evidently very profound, and from the character of the voice he thought the patient must have been almost, if not quite, a deaf-mute. He had a feeling that she was hearing more than a person in her condition might have been expected to hear.

Stenosis of Right Auditory Meatus.

By W. H. KELSON, M.D.

PATIENT, a girl, aged 17. Right auditory meatus represented by a sinus, into which a probe can be passed about 1 in., and which discharges a honey-like fluid. Mother states that the ear was severely injured at birth, and has discharged ever since.

Hearing (right side): Voice: 1. Conversational, 3 ft. } taken from
,, 2. Whisper, 6 in. } the side.
Watch $\frac{\frac{1}{2}}{30}$
Rinné—minus 10 sec.
Weber—right.

G_4 tuning-fork and high notes of Galton's whistle heard well. On inflation the air appears to pass into a cavity, but not through the sinus, and hearing is not improved thereby. Left side: Meatus and hearing normal.

DISCUSSION.

Mr. WHITEHEAD asked whether any microscopical or bacteriological examination of the fluid had been made, as it seemed important in regard to treatment. The causation was somewhat doubtful: it seemed as if it were secondary to chronic inflammatory trouble, and the question was whether anything further should be done now or whether it should be left.

Mr. LAWRENCE suggested that the meatus should be turned back and the condition behind inspected. The hearing could not be made much worse, and something might be found which would enable the condition to be remedied.

Mr. CHEATLE said he thought it was a case of chronic eczema of the meatus, and that improvement would take place on treatment.

Dr. URBAN PRITCHARD expressed strong agreement with Mr. Cheatle's observations. He thought it was a case of very chronic eczema, in which there was so much thickening that the meatus was almost closed. He asked whether any treatment for eczema had been tried, because that should precede any surgical means.

Dr. KELSON, in reply, said that no bacteriological examination had been made, but the microscopical one pointed to eczema, and there was very little pus in the discharge. It had been persistently treated as eczema, but it had come back as uncured. The treatment had extended over six months. The question was whether surgical interference was called for. There had never been any pain. When the discharge improved the hearing improved, but there was no permanency.

Two Cases of Epithelioma of the Ear.

By HUNTER TOD, F.R.C.S.

CASE I.

Primary Epithelioma of the Tympanic Cavity on the Left Side, with Secondary Infection of the Skin over the Mastoid Region.—Patient, a woman, aged 44. First seen August, 1906. History of otorrhœa for six months. A large perforation with granulations coming from upper posterior part of tympanic cavity. In spite of antiseptic treatment and frequent curetting away of granulations, healing not obtained. Ossiculectomy November, 1906. Further recurrence of granulations. February, 1907, complete mastoid operation. Auditory canal normal; no external signs of disease over mastoid process; no enlargement of glands. Cortex of mastoid sclerosed; antrum and mastoid cells found filled with granulations. Posterior wound closed, mastoid cavity being packed through the meatus. Rapid formation of granulations within wound cavity. Breaking down of post-aural wound with the formation of a sinus behind ear. In May, wound reopened; granulations curetted out and more bone removed. Malignant disease was suspected from the frequent recurrence and character of the granulations. Microscopic examination confirmed diagnosis of epithelioma. Consultation with Mr. Eve, who suggested tying of branches of external carotid, and at the same time removal of any infected cervical glands. Extensive operation by Mr. Eve in July. For some time afterwards growth seemed to diminish. Tympanic cavity and mastoid region now healed, but epitheliomatous ulcer the size of a halfpenny is situated over the site of the original post-aural incision. There is no further infection of the cervical glands in the anterior and posterior triangles of the neck.

CASE II.

Epithelioma of Right Auricle, with Secondary Involvement of Auditory Canal.—Man, aged 62. Warty growth noticed on lower part of auricle for some time. Microscopic examination confirmed diagnosis of epithelioma. Growth freely excised fifteen months ago. Patient did not again come under observation until recently. There is now scarring and perhaps recurrence of the growth on the auricle, with a large polypoid mass filling auditory canal and involvement of pre-auricular and cervical glands.

DISCUSSION.

Mr. WHITEHEAD said the first case was curious and remarkable in several features. The history of antecedent otorrhœa was very short. If carcinoma appeared in the middle ear there was a long history of antecedent otorrhœa, whereas if there were surface malignant disease starting in the auricle or in the auditory canal, it was almost invariably without any antecedent history of otorrhœa. The recurrence in the skin was remarkable, as the original disease was in the deeper parts.

Dr. MILLIGAN had usually found in such cases a history of very chronic otorrhœa; but two days previously he had operated on a case of epithelioma when the history was that the discharge from the ear had only lasted nine months. Cross-examination did not shake the patient in his statement. He had seen one or two cases before in which there had been suppuration for years.

Dr. BRONNER asked for further notes on the cases. He said that some years ago he showed before the Otological Society specimens of three cases, and one of the members said it was impossible. But when attention had been directed to the subject, it was found that such cases were not so very rare. In his experience they were nearly always fatal if they arose from the middle ear. There was a history of prolonged suppuration in most of them.

The PRESIDENT said that the first case interested him very much, and reminded him of one he saw many years ago in which the old Schwartze operation had been performed, leaving a sinus. There was still some discharge from the sinus, but an epithelioma developed, beginning at the lower margin of the sinus, and the patient died. In such a case nowadays operative measures would be tried, though he would not be very hopeful of the result. More recently he saw a case in which there was no history of otorrhœa. It was that of an old lady who came to him about two years ago with a very painful affection of the meatus. There was a slight thickening of the meatus, but more like a furuncle. He had a portion examined, and it was reported to be malignant.

He turned forward the auricle and removed the diseased part, with the post-auricular gland, which was enlarged; but the disease recurred and she died. As she lived away from Edinburgh he only derived information of the later stages from her medical man.

Mr. TOD, in reply, said that he had to be guided by the statement of the patient as to the duration of the otorrhœa. She had never been under any previous treatment. When he first examined her there was pus coming from the tympanic cavity, and granulations covered its upper and posterior part, but the auditory canal was not involved. He treated the case at first conservatively, curetting away the granulations twice under cocaine. As they recurred he advised removal of the granulations together with the malleus and incus. He did not, at first, think that the condition was anything more than an ordinary middle ear suppuration. Rapid recurrence of the granulations led him to advise the complete mastoid operation. The outer part of the bone was not affected, but the mastoid process was very vascular and filled with friable granulations, resembling the condition occasionally seen after scarlet fever. He did the complete operation, making a posterior meatal flap and closing the posterior wound with sutures. In spite of no apparent cause the granulations rapidly recurred within the mastoid cavity, and about the third week after the operation there was some swelling and redness at the lower part of the posterior wound, through which granulations began to protrude. The wound was reopened, the granulations curetted away and more bone removed. Owing to malignancy being now suspected the granulations were submitted to microscopic examination and found to have the character of a typical squamous epithelioma. A consultation was held with Mr. Eve (because, at the London Hospital, extensive operations on the neck, even if secondary to the aural affection, are considered to be within the sphere of the general rather than the aural surgeon). He (Mr. Tod) suggested removal of the petrous bone and of the cervical lymphatic glands. Mr. Eve operated, but did not consider it advisable to further touch the petrous bone. An extensive incision was made in the neck along the margin of the sterno-mastoid, which was exposed and cut across. Enlarged glands were removed from the posterior triangle. In the anterior triangle, owing to their adherence to the sheath of the vessel, it was necessary to remove part of the jugular vein. The branches of the external carotid artery were then exposed and tied. The mastoid wound was left open and packed with gauze. Gradually the anterior and tympanic portion became separated from the mastoid cavity, and eventually healed completely, so that, on looking into the auditory canal, a skin-lined cavity could be seen. With this the posterior wound became shallower and scarred over, with the exception of the formation of a small pimple at its lower margin. This seemed merely superficial. This small ulcer was removed by free incision; microscopic examination proved it to be epitheliomatous. The patient refused further operation. Since then the ulcer had progressed. There appeared to be no doubt that the present epitheliomatous ulcer was secondary to the original growth which began in the middle ear, now apparently completely free of disease.

Demonstration of Specimens.

By William Milligan, M.D.

(1) Photographs of two patients suffering from double facial paralysis.—Case 1: Male, aged 45. Left-sided suppurative middle ear disease since age of 7; admitted to hospital with complete facial paralysis upon the left side, mastoid fistula and post-pharyngeal abscess communicating with the ear, and atrophy of left facial muscles. Developed ear disease and right-sided facial paralysis. Paralysis on the right side recovering under treatment. Case 2: Male, aged 37. Syphilis contracted seven years ago. Pachymeningitis; paralysis of seventh and eighth nerves upon both sides.

(2) Temporal bone from a case of temporo-sphenoidal abscess with diffuse septic encephalitis of temporo-sphenoidal lobe; hæmorrhage into the lateral ventricle.

(3) Sequestrum from a case of labyrinthine suppuration.

(4) A protector for the facial nerve for use during the performance of labyrinthine operations.

(5) A series of lantern slides illustrative of the anatomy of the middle ear and labyrinth for teaching purposes.

(6) Temporal bone from a case of fracture of the base of the skull.

The President said that Dr. Milligan's preparations and slides were most beautiful, and they illustrated anatomical facts which, while perhaps not calculated to invite argument, were most valuable and instructive.

Otological Section.

February 8, 1908.

Dr. PETER McBRIDE, President of the Section, in the Chair.

Histological Preparations of the Human Cochlea.

By SYDNEY SCOTT, M.S.

METHOD of preparation (preliminary communication): Temporal bone from newly born child. Fixed in Zenker's fluid. Decalcified in nitric acid. Imbedded and cut in paraffin. Sections show the bony capsule of the labyrinth and tympanum, with the modiolus and lamina spiralis, the auditory nerve, spiral ganglia, and terminal nerve fibres in the organ of Corti. The organ of Corti is shown in tangential and radial vertical sections. The inner and outer hair cells can be seen, with "hair" fibrils; also the cells of Deiter, Hensen and Claudius. The rods of Corti are distinct with the tunnel of Corti and canal of Nuel in cross-section. Reissner's membrane is intact and membrana tectoria is well preserved. The sections show the structure of the ligamentum spirale and of the stria vascularis.

Staining methods: Hæmatoxylin, eosin, van Gieson's fluid, carbo-fuchsin, acid fuchsin, methyl blue, orange G.

DISCUSSION.

The PRESIDENT (Dr. McBride) expressed the thanks of the Section to Mr. Scott for bringing the beautiful preparations. Such work was very difficult to carry out, but by examination of the cochlea after death much useful information had been gained as to chronic deafness.

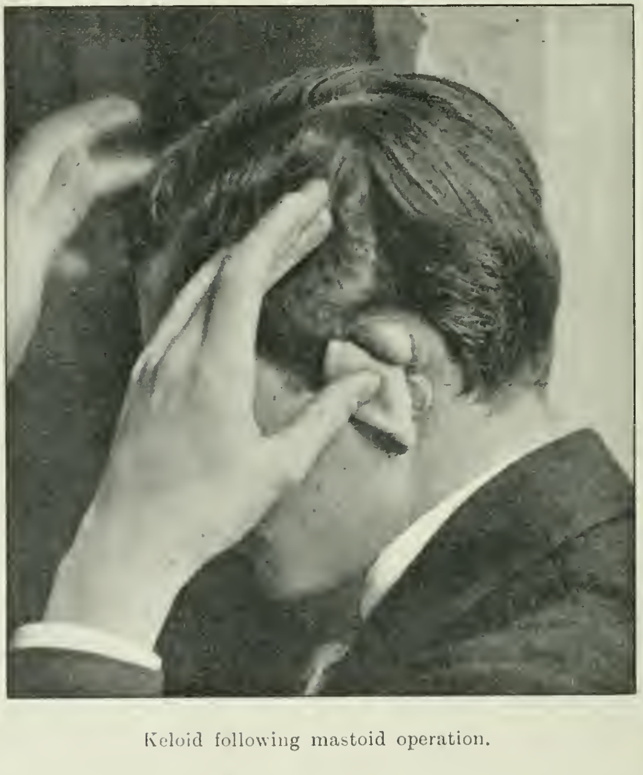
Mr. A. CHEATLE asked how soon after death the preparations were obtained.

Mr. SCOTT, in reply, said that particular case he got two hours after death. In some the time was ten hours, and up to that time the organ of Corti could still be seen by the method he had adopted.

Case of Keloid following Operation for Acute Mastoid Suppuration.

By HERBERT TILLEY, F.R.C.S.

PATIENT, male, aged 17. Two years ago the patient suffered from influenza, which was complicated by acute mastoid suppuration on the left side. Through the usual postaural curved incision the antrum was freely opened and drained. Only the upper half of the incision was sutured at the time of operation, the lower half was left open for purposes of drainage. Warm boracic fomentations were applied every three or four hours for the first two days until the acute inflammation and œdema of the soft parts overlying the mastoid had subsided.



Keloid following mastoid operation.

The patient made an uneventful recovery from his acute symptoms, although the exhibitor has no knowledge of any details of the after treatment because these were carried out by the patient's usual medical attendant, who has since died. When seen two months ago an excessive but irregular growth of keloid was seen to have developed in the whole line of the scar. It is most prominent in the upper half of the scar, where there is a smooth, ovoid, soft tumour the size of a small walnut, which is slightly painful on firm pressure. The patient's general health is excellent, his hearing normal and the tympanic membrane is intact.

DISCUSSION.

Dr. MILLIGAN said he had never seen so marked a case. There were three methods of treatment available: (1) Subcutaneous injection of fibrolysin, (2) internal administration of thiosinamin, (3) entire excision. He favoured the last. An interesting point was the method of development of keloid masses and scars. He had seen them in cases where the thyroid had been removed, and wondered whether such formations had anything to do with the mobility of the tissues.

Mr. WEST considered there were two main factors in the causation of surgical keloid. One was continued irritation of the wound, and the other, making the wound along the line of traction. It was seen in connection with badly planned scars in the neck after removing tuberculous glands. He did not think traction on the tissues entered into the present case, but a tube appeared to have been kept in longer than necessary, for as soon as the tube was removed the wound healed. He thought the result of excision would be quite satisfactory, and that there would not be recurrence.

Dr. DONELAN said he saw a case last year. The patient was operated upon in Paris in March, 1907, and a drainage-tube was left in, exposing the wound to infection for a considerable time, and on coming to England she developed a small keloid in the cicatrix. He excised that on August 30, and she has remained free from recurrence for now over six months.

Mr. FAGGE said that Mr. West's remarks were founded on his experiences of ear surgery only, but we must remember that in other parts of the body, as for instance in operations for appendicular abscess, prolonged drainage with a rubber tube did not lead to keloid. In his (Mr. Fagge's) opinion it was doubtful whether continual irritation was a factor; mastoid wounds, even if the ear was apparently clean, often failed to unite primarily, and yet keloid was a very rare result. He would rather confess that he did not understand the causation of keloid than account for it in a way which must be unsatisfactory, because it was not according to experience. In one case of keloid after a radical mastoid he excised it freely and carefully sutured the wound to obtain very accurate apposition, but it returned. There might be something in the position of the scar which made the difference. He had often noticed that in the ordinary oblique incision for the radical cure of inguinal hernia the lower part of the scar which involved the skin, over which the pubic hair grew, became hypertrophied, whereas the upper part of the scar was smooth. That was opposed to Mr. West's suggestion, because the upper part of the scar would be mobile and subjected to stretching, the lower part being fixed and much thicker.

Mr. WAGGETT agreed with Mr. Fagge's remarks. He did not think irritation by a tube had anything to do with it, nor had the mobility of the part, because after removal of tuberculous glands in the neck the wound might heal by first intention and yet keloid develop, and on removal of that keloid there was no return of it, though the mobility of the part was the same as that which existed after the first operation.

Dr. JOBSON HORNE considered an important point in the etiology was the situation of the wound. The largest and most troublesome keloid he had seen was when he was a student. It arose after an amputation of the breast, and persisted in spite of repeated operations. It was over bone, as in the present case, and the skin was stretched. Mr. Tilley, for reasons given in the précis of the case, had not referred to the post-operative treatment as a factor.

Mr. MACLEOD YEARSLEY said he had seen one case of keloid which was greatly benefited by X-rays, and suggested that they should be tried in this case.

Mr. C. A. BALLANCE said Dr. Nicholas Senn, whose lamented death was recently announced, had written on keloid some twenty-five years ago, in which he showed that on microscopical section, and, he thought, on cultivation also, there were included organisms which were retained during the process of healing. In certain cases no doubt the keloid scars were caused by the irritation of included micro-organisms along the line of the healing process. Many years ago he saw a case of spontaneous keloid with Sir James Paget. Sir James said that, in his experience, keloid tumours could be divided into two groups. Spontaneous keloid was a tumour which was locally malignant, because it returned always after removal, and therefore it should be left alone. The other group (keloid associated with wounds) sometimes spontaneously disappeared, and occasionally could be successfully removed by operation. He (Mr. Ballance) did not know whether the present case could be cured by operation. If the case had been his he would attempt to carry out wide excision in the hope of preventing a return.

Mr. SCOTT suggested that the growth be removed. Mr. Tilley should attempt to isolate the specific micro-organism, and, if possible, obtain an anti-vaccine from it.

Mr. HERBERT TILLEY, in reply, said that he could not give any details about the after treatment as this was superintended by the patient's medical attendant, who had since died. At the time of the operation the tissues over the mastoid were inflamed and œdematous, and pus was found beneath the soft tissues when these were incised. The outer antral wall was removed, free drainage secured, the upper half of the wound sutured, and warm boracic fomentations applied for forty-eight hours, till the congestion of the soft tissues had diminished. He did not think the tube was the cause of keloid, because this condition often appeared where no drainage-tube had been used, and he had recently seen a large keloid scar in the incision made for appendectomy where the wound healed by immediate union. Probably the patient's friends would prefer a trial of X-rays as suggested by Mr. Yearsley. He (the speaker) thought that if excision were attempted it might be wise to leave the wound to granulate and then apply a skin graft, a suggestion which had been made to him by Mr. Godlee, of University College Hospital.

Unusual Case of Cerebellar Abscess.

By D. R. Paterson, M.D.

Boy, aged 11, ill three weeks with high temperature; pain behind left ear and occasional vomiting; no discharge, hearing only slightly impaired on left side; tenderness along posterior border of left mastoid; optic neuritis in early stage; at operation antral mucous membrane swollen and granular; periosteum thick and fleshy, and bone soft and gelatinous-looking; tympanic cavity not touched. At completion of operation discharge noted in meatus for first and only time. Five days later, middle fossa, sinus groove, and posterior fossa by way of posterior antral wall were explored with a negative result. One week later, as general condition worse, soft bone was followed back; $\frac{3}{4}$ in. behind found small fistulous opening with track through dura, and quantity of thick odourless pus evacuated; probe passed downwards and slightly forwards; optic neuritis had become intense, with retinal hæmorrhages. Recovery uneventful without impairment of hearing. Temperature ranged from 99° F. to 103·4° F. Pulse, 80 to 104.

DISCUSSION.

Mr. A. Cheatle asked whether the pus was in the cerebellum or whether it was an extradural abscess.

Mr. A. L. Whitehead said one or two similar cases had been reported, and in one under his own care there was apparently nothing wrong with the middle ear and no diseased bone was found. It was a cerebellar abscess and the patient ceased to breathe during the operation and shortly afterwards died. He wondered whether it was possible for cerebral or cerebellar abscess symptoms to arise which would necessitate operation in a case where all the temporal bone disease had been removed and the wound had become dry and healed for some time. He had not come across such a case nor seen one recorded, and asked whether other members had.

Dr. PATERSON, in reply, said Dr. Permewan's case apparently occurred without perforation of the membrane. In the present case the notes said there had been no history of discharge, but directly after the operation there was some discharge in the meatus for the first and only time. It is possible, therefore, that discharge may escape notice in such cases. In answer to Mr. Cheatle the pus was certainly inside the posterior fossa of the skull, because one could pass a probe from the opening forwards and almost touch the dura, which had been previously laid bare from the antrum.

Right Temporal Bone of a Man, aged 42, showing how Pus may reach the Neck without passing through the Mastoid Process.

By ARTHUR H. CHEATLE, F.R.C.S.

A LARGE cell is present in the outer wall of the antrum, and from it cells pass downwards and inwards internal to the mastoid process and invade the occipital bone and reach the digastric fossa, the wall of which is thin and translucent. The lateral sinus bounds the lower cells behind, and the lower partition is very thin.

Ménière's Disease in a Girl, aged 14.

By RICHARD LAKE, F.R.C.S.

D. S., FEMALE, aged 28, seen on January 2, 1908. Her sister, who was a few years older, gave the following history: The patient had scarlet fever when aged 3, followed by right suppurative otitis media, which lasted for some length of time. The exact date at which cessation of the discharge occurred was not known, but she became completely deaf on that side before she was aged 14, for some time previously suffering from severe pain in that ear. One morning when patient was aged about 14, she was as usual awakened by her sister. On attempting to sit up she found that she was so giddy that this was impossible, and she was also unable to hear any sounds at all, even in the left ear, this, as already intimated, being the only one in which she previously possessed any power of hearing. The giddiness, which was accompanied at times by sickness, persisted for between two and three weeks, after which time she recovered her power of equilibrium, but not that of hearing. A careful inquiry failed to elicit any history of accompanying or recent disease, though there seemed to be some ground for suspicion that she might have been rather anæmic at the time.

On questioning the following further information was obtained: That, as far as one could judge, she could hear her own voice to a slight extent: that she had suffered from severe tinnitus since the time of the attack of fourteen years ago, and that she had lately had a slight recurrence of otitis on the left side. The patient also gave this extraordinary piece of information, that very occasionally she could suddenly and for a short time only hear certain high-pitched sounds. When this short interval of ability to hear is over she is prostrate, and suffers the greatest sensation of fatigue.

Examination of the Ears.—The membrana tympani and ossicles were entirely absent on the right side, while on the left the membrane showed no changes, and the malleus was mobile. On testing left ear it was found that by aerial conduction she was able to hear the tuning-forks C^2, C^3 and C^4, though only when vibrating strongly. She had a loss of only fifteen seconds on the mastoid to the C tuning-fork, and the highest note that she could hear by Galton's whistle (Galton-Edelmann) was C^5 (4,138·44 vibrations), the next being B^4 (3,906·17 vibrations).

I draw attention to a few points. The first is the age at which the patient was seized with an apparently genuine attack of Ménière's disease, an attack in which all the cardinal symptoms were present, and which must have been caused by a hæmorrhage into the labyrinth, whatever the cause of the hæmorrhage may have been. One has somewhat analogous cases of cerebral hæmorrhage in children. Here the greater destruction of the parts is in the posterior half of the labyrinth, and the cochlea, although rendered unserviceable, is not completely destroyed, as was shown when a sufficiently strong impulse is brought to bear upon the terminal filaments of the auditory nerve. Thus, for example, high air sounds of sufficient intensity to easily pass through solid and other matter were perceived by air conduction, whilst 2C, 1C, CC^1 and C^2 were all perceived by bone conduction. One feels able to make the statement definitely that the latter were all heard and not felt, as the patient was an extremely intelligent young woman—a pupil teacher, despite her misfortune.

DISCUSSION.

Mr. CHEATLE said that if the giddiness was not due to suppuration he would suggest that the Ménière's symptoms were due to congenital syphilis. Some years ago he saw a boy who was stone deaf in one ear, and he gave a history of having been sick and giddy for a week. No other signs of congenital syphilis were found, but three months later the patient saw Dr. Pritchard, who found tertiary ulceration of the pharynx. There was no doubt that the Ménière's attack which he had, lasting a week, was due to congenital syphilis. He thought the present case should be carefully examined for signs of syphilis, especially for choroiditis.

Mr. WEST suggested the following interpretation of the case: The patient had chronic right suppuration, and when aged about 14 she rapidly lost hearing in that ear because the labyrinth of it was involved by carious labyrinthitis and then by acute vestibulitis in that ear, causing vertigo, and leaving her totally deaf on that side. It was unfortunate that Mr. Lake had not given hearing test and stability test results on the right side, and especially a comparison of the two sides. It was possible she had never had good hearing on the other side. A personal friend of his was a case exactly similar. The lady was deaf for useful purposes on one side with chronic catarrh, and she used to hear with a chronic suppurating ear. Some years ago she became totally deaf and had vertigo and tinnitus, was sick and giddy, and unable to stand up for some days. On her "good days" she still heard certain high-pitched sounds, and referred them to the ear which became deaf from labyrinthitis. Tinnitus persisted. He had never heard her say she experienced prostration when she heard. That point raised the question of hysteria in the case. He would be glad if the further particulars could be furnished at some time, and also in which ear she seemed to hear her voice and the effect of rotation on the stool.

Dr. DAN MCKENZIE agreed with a previous speaker in ascribing the onset of the Ménière's symptoms to the pre-existing suppuration. One feature in the case which supported this view was the fact that there was no mention in the history of a recurrence of the vertiginous attacks.

Mr. C. H. FAGGE supported Mr. Cheatle's view that the condition was due to congenital syphilis. He brought forward a similar case at one of the earliest meetings of the Otological Society[1] which several members suggested was due to congenital syphilis. There was no confirmation, except limited choroiditis in one eye. At the time it was treated as hysteria, but his own view had been that it was early Ménière's disease.

Mr. LAKE, in reply, said the patient lived in Nottingham, and as she was stone deaf it was very difficult to examine her; moreover, he could not devote a good part of the day to one patient. The result of his enquiries was that he thought syphilis could be excluded as far as that was possible. He could not trace any hearing in the old suppurating (the right) ear. He thought Weber's test usually of little value; here of course it would have been. Ménière's classical case had only one attack of vertigo.

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[1] *Trans. Otol. Soc.*, ' . p. 59.

A Case for Diagnosis.

By R. Sturgeon Cocke, F.R.C.S.Ed.

The patient was a girl, aged 8. There is a hard swelling about half the size of a pigeon's egg situated on the left mastoid process. This when first noticed by the child's mother fifteen months ago was about the size of a pea. It is not translucent, and the skin is freely movable over it. The swelling has appreciably increased during the last three months. The auditory canal and tympanic membrane are normal, and there is no deafness.

DISCUSSION.

The President said that in discussing the case in the other room there seemed to be a difficulty in concluding whether the growth was movable or not. He was not prepared to commit himself on the point. It seemed to be a bony growth, but he did not know whether it was continuous with the subjacent bone. If the patient would consent it seemed right to cut down on it and remove it.

Dr. Jobson Horne said that the tumour on careful examination appeared to be mobile, and that observation was supported by the history given on the agenda paper. He advised removal.

Mr. Cheatle felt certain that it was movable. He would hazard the diagnosis that it was a calcified gland.

Dr. Pritchard said he had a similar condition behind his left ear. At first he thought it was bony or cartilaginous, but his final conclusion had long been that it was a gland. It was slightly movable on the mastoid. His own differed from the case shown in that it had not increased in size; but if that in the case shown continued to grow he thought it should be removed and examined. He would be much interested in the result.

Mr. West did not consider it was a gland. It was not a bony tumour either, and it moved. He regarded it as either enchondroma, endothelioma, or a solid dermoid.

Mr. Waggett had at first formed the opinion it moved $\frac{1}{8}$ in. in a lateral direction, but later, by putting his hand on the tip of the mastoid, he had convinced himself that it was absolutely fixed. It was bony.

Mr. Fagge said it was mobile, and was either a connective tissue tumour, such as an endothelioma, or a calcified gland. But in the absence of any condition, either in the head, on the scalp, or in the meatus, which could be a cause for a calcified gland, and in the absence of other enlarged glands, he inclined to the alternative of a new growth.

Mr. Whitehead agreed that it was mobile, and thought it was a calcified gland.

Mr. Kelson said the tip of the tumour bent, but it did not move bodily.

Dr. W. HILL thought the suggestion that it was of an enchondromatous nature was probably right.

Mr. SCOTT thought it was a calcified gland, and that it was fixed to the outer surface of the periosteum.

(On operation on February 11, 1908, the growth was found to be absolutely immobile, and was apparently a cancellous osteoma. The bone was hard externally, but internally was more cellular. It was freely removed by chiselling round its edge.)

A Case of Vertigo, the Symptoms pointing to some Inflammatory Lesion within the Semicircular Canals.

By HUNTER TOD, F.R.C.S.

J. S., aged 26, a potman. Had discharge from right ear for over ten years. Four years ago I performed the complete mastoid operation, and, owing to the constant recurrence of granulations on the inner wall of the tympanic cavity, the wound had to be subsequently reopened and, in addition, curetted four times under gas anæsthesia. Eventually complete healing took place.

The patient was admitted to the hospital on January 18, not having been seen for over two years, owing to his having a fit whilst serving in the bar. On admission, he gave a history of feeling ill during the last month and of having pain behind the right ear and down the side of the neck. There had been frequent attacks of giddiness, during which objects seemed to pass from below upwards. For the last fourteen days there had been constant singing in the right ear. When admitted there was a foul discharge from the right ear. On examination, a large plug of cotton wool was discovered within the ear and was removed. It had apparently been inserted some months previously and been forgotten. On cleansing the ear, the cavity was found to be lined with epithelium, but there was no sign of any granulations nor fistulæ of the bony walls suggesting a lesion of the labyrinth. The eyes were normal, but there was marked lateral nystagmus, increased on looking to the right. Hearing tests showed marked deafness; the watch could not be heard, even on contact; whispering only 6 in. off; the high and low tuning-forks were badly heard by air conduction; bone conduction was comparatively well heard, and Rinné was negative, but Weber's test was indefinite. On syringing the ear there was marked vertigo and a feeling of nausea. Coördination tests—Static: When standing unaided, with the feet close together, there is an irregular swaying of the body. With the eyes closed there is a tendency to fall backwards or towards the affected side. Dynamic: With the eyes open, the patient can walk fairly steadily, but if he attempts to turn sharply the giddiness is so great that he has to clutch objects to prevent falling. With the eyes closed, he falls to the affected side. Jumping tests are impossible owing to instability.

On admission to the hospital the diagnosis, at first, was thought to be that of internal ear suppuration, but, owing to the patient being somewhat under the influence of alcohol, no reliance could be placed on the hearing and coördination tests. Against internal ear suppuration was an even temperature, with ability to hear high tuning-forks, and the existence of bone conduction on the affected side. Also, after thoroughly cleansing the ear, no definite lesion of the inner wall of the middle ear could be discovered. The symptoms, therefore, seem to point to some irritation of the semicircular canals, perhaps due to the septic plug of cotton wool which had been left in the ear. The patient has been taking a mixture containing potassium iodide and bromides, and he certainly seems very much better.

DISCUSSION.

Mr. A. L. WHITEHEAD said he did not understand where the pus was coming from. Mr. Tod said in the notes that a large plug of wool was removed, soaked in pus. After the ear was cleansed, was everything perfectly healed and dry?

Mr. SCOTT remarked on the statement that objects had seemed to pass from below upwards, a direction which was not usual in cases subsequently found to be lesions of the external or superior canals. Then there had been marked increase in nystagmus on looking to the right side, *i.e.*, the affected one. In most inflammatory conditions of the labyrinth, however, the nystagmus was more marked in the opposite direction. The patient heard comparatively well by bone conduction, which was rather against intralabyrinthine lesion. He noticed at the present time that the patient had as much tendency to fall to the left side as to the right. If the lesion in this case was labyrinthine he thought it must be extralabyrinthine, that is, due to irritation from without. The strong element of neurosis greatly increased the difficulty of diagnosis.

Mr. LAKE said he did not see the case, but in the last two cases of suppurating labyrinth which he had there was relatively good hearing. In most cases where there was suppuration in the posterior half of the labyrinth the anterior half of the cochlea was not involved, nor was it except where there was necrosis affecting the whole.

Mr. WEST said it would be well if the results of putting the patient on the rotation stool could be known with regard to the direction of the nystagmus after rotation to the right and the left respectively, with the eyes closed during the period of rotation.

The PRESIDENT asked whether Mr. Tod had tried syringing with hot and cold water, or what his views were on that test, as employed by Barany.

Mr. TOD, in reply, said the patient did not present the same clinical features as he did a fortnight ago. The symptoms were then so urgent that he (Mr. Tod) was sent for in order to operate, the diagnosis being given as that of internal ear suppuration. In answer to Mr. Whitehead, Mr. Tod said that although on admission there was a foul discharge in the right ear it was apparently largely due to a plug of cotton wool which had evidently been lying in the ear for a considerable period. On its removal there was no sign of any granulations or fistulæ of the bony walls suggesting a lesion of the labyrinth, and for this reason the diagnosis of internal ear suppuration was questioned and operation postponed. As the suppuration ceased as a result of treatment within two or three days, and the cavity of the ear was found to be lined with epithelium, true internal ear suppuration was excluded. Mr. Tod therefore agreed with those who considered that the symptoms were probably due to external causes, presumably from irritation from the cotton wool plug. In answer to the President he said that there was marked giddiness on syringing with very hot or very cold water, the import of which was doubtful. He would, in addition, be pleased to carry out von Stein's tests as suggested by Mr. West.

Lateral Sinus Thrombosis ; subsequent Meningitis (Meningitis serosa) ; recovery.

By HUNTER TOD, F.R.C.S.

F. S., A boy, aged 8, was admitted to my out-patient department in May, 1907. There was a history of otorrhœa on the right side for a period of nine months. Pus was seen to exude from a marginal perforation situated in the posterior part of Shrapnell's membrane and the upper posterior quadrant of the pars membranosa; the rest of the tympanic membrane was indrawn and somewhat congested in its posterior portion. There was extreme deafness; the watch could not be heard, the voice only in close proximity to the ear, and the tuning-forks were badly heard by air conduction, although well heard by bone conduction. As there was no improvement, in spite of conservative treatment for six weeks, and as there were occasional attacks of headache and pain in the ear, ossiculectomy was advised. This was performed on June 25. At the time of the operation it was found that the disease was more extensive than was at first supposed. The complete mastoid operation was therefore performed on July 2. The cortex was thin but sclerosed, the antrum and mastoid forming a large cavity lined with epithelium, and filled with a soft, pultaceous, putrid mass ; the outer wall of the lateral sinus was lying exposed within the cavity. The posterior wound was not completely closed. On July 5 (three days after the operation) there was a definite rigor. On removing the gauze hæmorrhage took place from the lateral sinus, but was easily controlled by packing. On July 10 a second rigor occurred. On July 11 there was marked pain in the right hip, causing the patient to resist movements of the limb. The temperature varied between normal and 101° F. There was some stiffness of the neck, the head being kept in a fixed position. The optic fundus was normal. On July 12 the wound was reopened and more bone removed, the sinus being freely exposed above and below the point at which bleeding took place. A plug of gauze was inserted between the outer surface of the sinus and the bony wall of the skull, so as to completely obliterate its lumen. The outer wall of the sinus was then freely slit up with a knife. There was considerable bleeding from the superior petrosal sinus. The outer wall of the sinus over the affected area was markedly thickened and friable, the thrombus appearing partial rather than complete. For the next few days the patient seemed more comfortable, the temperature gradually falling from 101° F. to normal. On July 23 left facial paralysis was noticed and rapidly became complete. Knee-jerks not obtained. There was no paresis of the ocular muscles, no headache, and no vomiting. On July 29 some swelling of the right optic disc was noticed for the first time, the left optic fundus being normal, and with this there was paresis of both external recti muscles, which gradually became more marked on the left side. During the next few days there were attacks of vomiting, tendency to drowsiness, headaches, and disinclination to take food, the patient becoming markedly emaciated. There was still stiffness of the neck, but no marked retraction. On August 8 the facial paralysis began to diminish, with complete recovery four days later.

For a few days the general condition remained the same, but was no longer accompanied by headaches nor vomiting. There were no fits, and the mental condition remained good. Gradual improvement took place, and by the end of August he was sufficiently well to be allowed up. The right external rectus was now normal, but the left completely paralysed. Ophthalmoscopic examination of the eyes showed that the optic neuritis on the right side had practically cleared. The patient was shortly afterwards made an out-patient, but I did not see him again until the first week in October. His mother said he had occasional attacks of headache, but beyond general weakness had been otherwise well. At the beginning of October the eyes were examined by Mr. Lister, who reported subsiding optic neuritis on the right side with optic atrophy on the left. A further report from Mr. Lister, on December 6, says: "On the right side there is no longer swelling of the optic disc; on the left side there is definite optic atrophy."

Except in the early stage there was no pyrexia, the temperature being on the borderland of normal with an occasional rise to 100° F.; the pulse varied between 80 and 100, and seldom fell below 70.

The diagnosis of internal ear suppuration was excluded owing to the absence of vertigo and the persistence of bone conduction. The involvement of the sixth nerve on both sides, with the seventh on the opposite side; the optic neuritis on the affected side with subsequent optic atrophy on the opposite side, together with the general condition of the patient, suggested that the symptoms were due to a non-suppurative meningitis.

Case of Pulsating Growth in the Left External Auditory Meatus.

By J. Barry Ball, M.D.

The patient, a man, aged 73, first became aware of something wrong in the left ear about ten years ago. He consulted Dr. McBride in Edinburgh, and learnt that he had a small growth in the meatus. As far as he can tell it has increased slowly and gradually since that date. It has caused him no discomfort, beyond deafness in the left ear, until the last two months, when it began to bleed. It has bled several times slightly, and on two occasions rather freely. There is no history of any discharge or other ear trouble before the growth began. He has felt a distinct beating in the ear for the last four or five months, perhaps longer. The growth fills the meatus and protrudes slightly from the orifice. It is of a pale red colour, and the free surface is slightly eroded. There is distinct expansile pulsation in it and the pulsation is communicated to the auricle and the region in front of the auricle. The attachment is deep in the meatus, but has not been exactly made out as manipulation causes hæmorrhage. In view of the attacks of hæmorrhage, and possible trouble from this symptom, suggestions as to the best method of dealing with the growth, which is presumably angeiomatous in nature, are invited.

DISCUSSION.

The PRESIDENT said he believed he saw the patient ten or twelve years ago. He believed he then had a growth about as large as half a pea on the floor of the meatus. It looked cystic, and the bleeding resulting when he punctured it was so severe that it was difficult to check it with sterilised wool, which was all he had handy. He had not seen another case like it, nor seen the record of one. It was an angeioma in the wide sense of the term. When he first saw the tumour he had an idea that it might spring from a displaced vessel, *e.g.*, jugular.

Mr. WAGGETT said a similar case was sent to him by Dr. Law for mastoid operation. He did not quite remember the details of the history. The patient was a healthy woman, aged 25, and the tympanum was occupied by a very tough vascular growth, so that with the spoon it could not be easily got away. On elevating it from below there was such a spout of blood that, for a moment, he thought he had opened the carotid. In subsequent dressings there were excessive hæmorrhages. His impression from microscopic examination was that it was sarcoma. He opened the wound a fortnight later and removed the growth very freely. He could not, however, satisfy himself of the complete character of the operation on account of the hæmorrhage, and he had intended to do a third operation. Perfect healing occurred, nevertheless, and three years after the operation the scar looked exactly like that after a normal mastoid operation.

Dr. MILLIGAN thought the duration of the disease was against malignancy. He would suggest putting a temporary ligature round the external carotid and noting the effect. If that were not successful, the wound should be enlarged and direct compression of the external jugular vein made. Such a growth was fraught with danger, because of hæmorrhage, which might come on at a time when the necessary remedies were not available. Although the patient was aged 73 he thought that it would be quite justifiable, from the history given of repeated and severe hæmorrhage, to make such an exploratory operation.

Mr. FAGGE said that, as the patient had had the swelling ten years, and it was not appreciably worse, there seemed no need for surgical interference. The occasional bleeding was easily controlled, and what was clinically known as malignancy could be excluded, though he believed that, microscopically, such tumours could not be distinguished from sarcoma. But, clinically, this tumour was not malignant, and he counselled leaving it alone.

Dr. W. HILL thought it should not go out that the Section supported the proposal to tie the carotid in a comparatively trivial case like the one under discussion. He had had, and had seen, undesirable results from tying the carotid.

Dr. BARRY BALL thanked members for their suggestions, which were mainly in the direction of doing nothing, and that had been his own feeling, on account of the man's age and because he was not a very healthy subject. A year ago he had what appeared to be a slight cerebral hæmorrhage. He did not think the bleedings from the growth had been very bad so far.

A Case of Primary Epithelioma (?) of the Tympanum following Chronic Suppurative Otitis media.

By A. L. WHITEHEAD, B.S.

THE patient, a man, aged 28, had enjoyed good health, with the exception of a chronic, painless, purulent discharge from the right ear, since infancy. There was no history of syphilis. In September, 1906, a polypus was removed by his medical attendant, who noticed that the hæmorrhage after removal was unusually free and persisted for several hours.

Three months later, when he first came under my care, the auditory canal was filled with a large red polypoid mass which bled freely at the slightest touch with a probe. There was no hearing power on the affected side and bone conduction was lost, but on the left side the membrana tympani and hearing were normal. The nose and nasopharynx were healthy. The polypus was removed with the snare and the base, which seemed to lie upon the promontory, was scraped. There was severe hæmorrhage, arrested by pressure.

Six weeks later he returned with the auditory canal filled with a mass of granulations which bled profusely when touched. No pain had been experienced and the general health was good. A radical mastoid operation was performed; the bone seemed abnormally vascular and the antrum and mastoid cells contained pus and ordinary granulation tissue. In the middle ear a large fleshy mass was found, from which copious hæmorrhage occurred when scraped. The growth extended deeply into the cochlea and petrous bone and forwards, involving the orifice of the Eustachian tube. The pharyngeal end of the tube was examined and found to be healthy. The growth was curetted away as thoroughly as possible, the hæmorrhage, however, being almost alarming and as profuse as that which occurs when the sigmoid sinus is opened. It was arrested by pressure. Some paresis of the facial nerve followed the operation. For about three weeks free bleeding took place from the middle ear each time the wound was dressed, otherwise healing was of the usual character. The skin wound healed by first intention and the cavity was dry and covered with epithelium in eight weeks after the operation.

There has been no recurrence of the disease up to the present, that is, about twelve months after the operation. The pathologist's report is that the growth has the structure of an atypical carcinoma: numerous slender tongue-like processes of epithelial cells separated by fibrous tissue invading the tissue in all directions. The surface epithelium is unaltered. The meatal walls are quite free from disease.

DISCUSSION.

Mr. WAGGETT suggested that Mr. Whitehead should allow the Morbid Growths Committee to have the specimen on account of the divergence of opinion as to the microscopical appearances of malignant disease and of the history of cure after what was presumably not a complete removal of the petrous bone.

Dr. MILLIGAN said the paper opened up the question of the frequency of epitheliomatous disease of the middle ear. He thought most of the cases seen were cases of extension from the external ear. A suggestion was made that the Section should have a symposium on rare diseases, and this, he thought, would be a fitting subject. One case he saw some years ago had carcinoma starting in the mucous membrane of the middle ear, extending deeply into the petrous bone and eroding the internal carotid with fatal results. Post mortem there was very extensive middle ear disease, internal ear disease, erosion of carotid artery, &c. He did not think that malignant disease starting in the external ear and extending into the middle ear was so very uncommon, but he thought primary epithelioma of the cavum tympani very rare.

Mr. TOD said, in reference to the frequency of malignant disease of the middle ear, that he had looked up the records of the London Hospital during the past ten years and found that no such case had been admitted with the exception of the one which he showed at the last meeting. As more than 200,000 patients were seen annually at the hospital this in itself was evidence of the rarity of the affection. There had, however, during this period been over thirty cases of malignant disease of the pinna, in the majority of which the whole or part of the auricle had been removed. What was the ultimate result of these latter cases he did not know, but could merely say that he could find no record of deaths from this cause in the clinical or post-mortem reports of the hospital.

Mr. CHEATLE said that carcinoma of the outer ear was not uncommon. From the history and further progress he doubted whether Mr. Whitehead's case was epithelioma. He (Mr. Cheatle) had mentioned one case of epithelioma in the middle ear in which there was no involvement of the meatus or pinna. There was paralysis of all the nerves passing through the jugular foramen, and some discharge from the ear. The middle ear was occupied by a mass of granulation tissue, and it was found to be epithelioma.

Mr. WEST said it was difficult to say whether a given growth was true middle ear growth or meatal growth: in both it seemed to be squamous-celled carcinoma. He believed practically all the cases started in the meatus. During the last twelve months he had operated upon two very early cases of malignant disease of the auditory meatus; both started in the deepest part of the floor. In one at a very early stage there was an elevated roll of tissue in the floor of the meatus with purulent discharge from the meatus, the membrane appearing normal; a little later this roll was ulcerating. At the operation there was partial sequestration and destruction of the tympanic plate. The growth had extended into the floor of the tympanum, though the greater part of the tympanum was free. In the other case the growth extended into the antrum and tympanum. In both cases he removed the whole tympanic plate and made a very free removal of the growth, including the whole of the cartilaginous meatus with the pre-auricular gland. Both cases had healed and the patients were doing well. Skilled pathologists had pronounced both specimens to be typical squamous-celled carcinoma.

Dr. PATERSON said that in one case the growth seemed to proceed from the promontory. He made a scraping there, and the report he got was that it was squamous-celled epithelioma. Arrangements were made for operation, but the patient died suddenly in a fit. He had been suffering from great pain in the head. The medical attendant made a post-mortem examination, and reported that the temporal bone was extensively infiltrated with growth. There was no trace of implication of the auditory meatus.

The PRESIDENT thanked Mr. Whitehead for his paper. He understood him to say "cases of carcinoma beginning in the meatus, with which we are all familiar." But that was contrary to his own experience and to his reading. In the cases in literature it was said the carcinoma had grown on the top of the middle ear suppuration, and no details were given as to the beginning of the growth. It looked almost as if Yorkshire was prolific in malignant disease, because Dr. Bronner, of Bradford, had brought forward a number of cases of malignant disease beginning in the meatus. Assuming it began in the meatus, was one to presuppose a pre-existing perforation of the tympanic membrane before it attacked the middle ear? or was it suggested that the growth perforated the membrane?

Mr. WHITEHEAD, in reply, said he had seen three cases associated with chronic suppuration, and he thought from that that the disease must be fairly common. In those cases there was prolonged suppuration, and then a growth sprang up deep in the meatus and extended inwards towards the middle ear and outwards through the auditory canal, involving the skin. Epithelioma of the ear seemed to be rare, except where it affected the pinna, and those might be common in other places besides Yorkshire. The other cases were a distinct class. Dr. Paterson's case seemed to be as clear as one could consider such cases to be. Mr. Cheatle doubted whether the present recorded case was epithelioma, and he (Mr. Whitehead) was inclined to agree with him; that was why he put the query into the title. He would gladly give the section to the Morbid Growths Committee, and if he could get further sections from the pathologist at Leeds he would send them up also.

Otological Section.

March 7, 1908.

Dr. PETER MCBRIDE, President of the Section, in the Chair.

The Operative Surgery of Labyrinthitis, based upon an experience of thirty cases.[1]

By C. ERNEST WEST, F.R.C.S., and SYDNEY SCOTT, M.S.

SYNOPSIS.

I.—INTRODUCTION.
II.—ANATOMY OF THE LABYRINTH.
III.—MORBID ANATOMY AND PATHOLOGY.
IV.—SYMPTOMATOLOGY.
V.—OPERATIVE SURGERY, WITH RECORDS OF CASES.
VI.—SPECIAL TESTS AND INDICATIONS FOR OPERATION.
VII.—RESULTS.
VIII.—BIBLIOGRAPHY.

I.—INTRODUCTION.

Deliberate and planned surgical operations on the auditory labyrinth for the relief of infective processes which have extended to it may be said to date from the publication by Jansen [14] of his classical cases at the Moscow Medical Congress in 1897. Much addition to our knowledge of the subject was made by Hinsberg [10] in an important paper in 1902, and, following this, attention was drawn to the subject in this country by Dr. Milligan [20] and Mr. Whitehead [38]. Since then the contributions to the literature have been numerous, and valuable papers have been published by von Stein [29],

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[1] Of which twenty-six were operated upon.

Brieger [3], Bourguet [2], Panse [25], Friedrich [8], Gradenigo [9] and Milligan. Our knowledge of the surgery of the non-suppurative conditions of the labyrinth has been extended by Mr. Lake [16], while numerous isolated cases of labyrinthine disease have been brought before the notice of the late Otological Society. We have ourselves been keenly interested in the subject during the last four years, and have at various times, separately, published accounts of individual cases [32], which will be again referred to in the present paper. We have felt of late that interest in and recognition of the importance of the surgery of the labyrinth has been rapidly increasing, and have ventured to offer you the substance of our experience and the results of a study of it. An accurate appreciation of the anatomy and relations of the bony labyrinth is so essential to its successful surgery that we make no apology for prefacing our paper with an anatomical description, and would merely say that we have mentioned no point which we have not personally examined and substantiated.

II.—Anatomy of the Labyrinth.

The bony labyrinth lies within the mass of the petrous, to the inner side of the chambers of the tympanum, aditus, and antrum. It is thus confined between the posterior, superior, and outer or basal surfaces of the petrous, and comes into close relation with each of these. For the operator it is hedged about by six structures of capital importance: the internal carotid artery in front of and below the cochlea, the meninges of the middle cranial fossa and the cerebrum above, the meninges of the posterior fossa and the cerebellum behind, the vault of the jugular bulb below and behind, and the facial nerve on its outer side. Its wall or capsule is formed of very compact brittle bone, which is clearly marked off from the surrounding more vascular and cancellous bony tissue; in the bone of the newly born this differentiation is so sharp that the labyrinth may be completely shelled out of its surroundings by the use of a blunt scalpel. The chambers of the bony labyrinth fall into two sections: a posterior, consisting of the vestibule and semicircular canals, and an anterior, consisting of the cochlea. The isthmus of communication between the two is a narrow one, so that inflammatory processes frequently become arrested and localised in one or other portion of the labyrinthine cavity.

The *vestibule* is an ovoidal cavity: it lies to the inner side of the postero-superior part of the promontory, the fenestra ovalis, which opens into its central portion, and the tympanic part of the facial aqueduct. On its antero-internal aspect it is separated by a very thin plate of bone from the posterior half of the fundus of the internal auditory meatus. Through apertures in this plate pass the branches of the vestibular nerve accompanied by their prolongations of the brain membranes and of the subdural and subarachnoid spaces. The dimensions of the vestibule are: antero-posteriorly about 6 mm.; from above downwards 4 mm. to 5 mm.; from without inwards 3 mm. The walls of the cavity pass into each other and cannot be sharply delimited. On the region corresponding to the internal auditory meatus (antero-internal wall) are two depressions: one rounded, lying below and in front, the recessus sphæricus, in which is lodged the saccule; one oval, lying above and behind, the recessus ellipticus, in which lies the utricle. These depressions are separated by an oblique ridge, the crista vestibuli. The floor of each of these

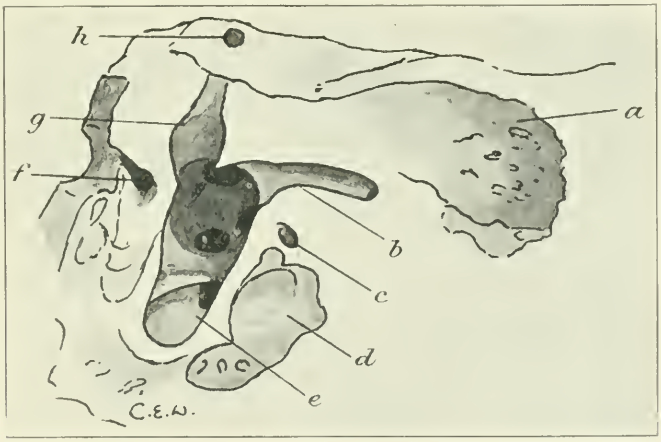

Fig. 1.

Cavity of Left Vestibule after removal of its antero-external wall.

(*a*) Mastoid antrum.
(*b*) Outer limb of external semicircular canal.
(*c*) Aqueduct of Fallopius at junction of second and third parts.
(*d*) Fossula rotunda and sinus tympanicus opened up.
(*e*) Commencement of scala tympani of cochlea, with fenestra rotunda.
(*f*) Termination of first part of aqueduct of Fallopius.
(*g*) Ampullary end of superior semicircular canal.
(*h*) Superior semicircular canal at commencement of inner limb.

Within the vestibule are seen the joint openings of the superior and posterior canals and the openings of the inner end of the external and lower end of the posterior canals; also the oblique opening of the aqueductus vestibuli.

depressions is perforated by a number of fine foramina, transmitting respectively the nerves to the saccule and to the ampullæ of the superior and external semicircular canals, while those to the utricle pass through perforations in the broadened anterior end of the crista vestibuli. The nerve to the ampulla of the posterior canal enters the vestibule on its inner wall, posteriorly and inferiorly, by a number of small apertures leading from the fundus of the canal of the foramen singulare close to the ampullary opening. On the inner wall behind the recessus ellipticus is the vestibular end of the aqueductus vestibuli opening obliquely, so as to form a gutter, directed forwards and downwards.

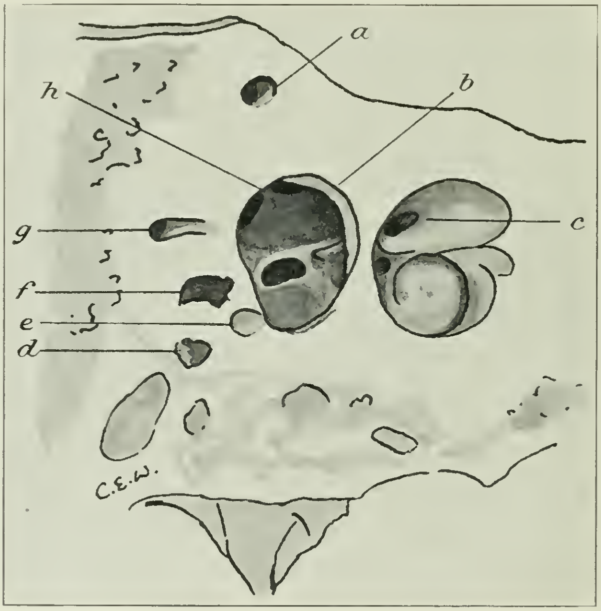

Fig. 2.

Cavity of Left Vestibule after removal of postero-internal wall, with part of fundus of internal auditory meatus.

(*a*) Superior semicircular canal lying below the superior arcuate eminence.
(*b*) Cavity of vestibule.
(*c*) Fundus of internal auditory meatus with opening of aqueduct of Fallopius indicated by the line.
(*d*, *f*) Parts of the sinus tympanicus opened by the section.
(*e*) Ampulla of posterior semicircular canal.
(*g*) Inner limb of external semicircular canal.
(*h*) Openings of outer limbs of superior and external semicircular canals.

In the roof of the vestibule are three openings: two externally, situated close to each other, for the ampullary ends of the superior and external semicircular canals; one internally, for the common opening of the inner crus of the superior and the upper crus of the posterior canals. In the posterior wall is one opening, that of the inner crus of the external canal. In the floor are two openings: one lying internally and posteriorly, for the ampullary end of the posterior canal; one externally and in front, by which the vestibule becomes continuous with the cavity of the cochlea. This opening is elliptical and largely floored by the shelf formed by the commencement of the lamina spiralis ossea in the floor of the vestibule, so that the vestibular cavity is broadly continuous with the scala vestibuli, while communicating by only a narrow slit with the scala cochleæ.

The outer wall of the vestibule corresponds with the following portions of the inner wall of the tympanum, viz., the tympanic part of the aqueduct of Fallopius, the pelvis ovalis, and the stout bar of bone which lies below it and stretches back from the promontory, cutting off the pelvis ovalis from the fossula rotunda. The floor of the vestibule lies at the level of the highest part of the notch of the fossula rotunda. The fenestra ovalis forms a wide defect in this wall, which is closed by the foot-plate of the stapes and the narrow annular ligament.

The *three semicircular canals* open, as has been described, into the vestibule at either end. They lie in planes which are approximately at right angles to each other: that of the external canal has a slight obliquity from the horizontal downwards and backwards, so that the upper face of this plane would look upwards and a little backwards and outwards: those of the superior and posterior canals are nearly vertical, that of the superior lying outwards and forwards at right angles to the superior border of the petrous, that of the posterior lying outwards and backwards and approximately parallel with the posterior surface of the petrous. The superior canal has, in addition, a slight but constant sigmoid curve, passing at first somewhat backwards in the upward course of its outer limb. The ampullæ open directly into the vestibule. Each of the canals has a definite and important relation with a surface of the petrous. The superior canal pushes up the superior surface into the arcuate eminence, which we shall call the superior arcuate eminence. In early childhood the compact wall of the canal is largely visible upon the upper and posterior aspects of the bone, arching over its superior border. In adult life there is, as a rule, an incrustation of bone covering, partly masking the arch of the canal; it is rarely more than $\frac{1}{16}$ in.

in thickness and is occasionally entirely absent on the upper surface. Rarely, in the macerated bone, there is the appearance of an elongated defect in the upper wall of the canal. We have never seen this in the infantile bone, and it may be an artefact. Under the arch of the canal in infancy the deep subarcuate fossa passes outwards and backwards, to come into close relationship with the upper part of the inner wall of the mastoid antrum; it is filled by a vascular plug of loose fibrous tissue, continuous with the outer aspect of the dura mater. A conspicuous vein is contained in this tissue, joining the superior petrosal sinus. One of us has also traced a vein from this area through an aperture in the

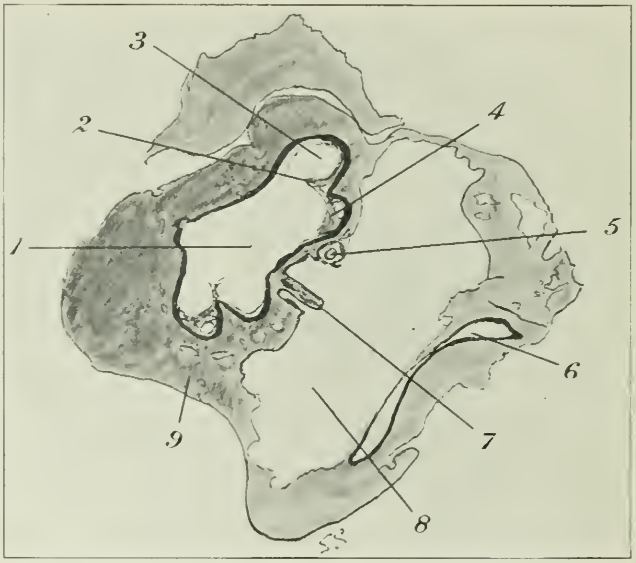

Fig. 3.

Vertical Coronal Section of Human Labyrinth, with cristæ of the superior semicircular canals.

(1) Vestibule.
(2) Crista of superior ampulla.
(3) Ampulla of superior semicircular canal above ampulla.
(4) Base of crista of external ampulla.
(5) Facial nerve.
(6) External auditory meatus.
(7) Posterior crus of stapes.
(8) Tympanic cavity.
(9) Crista of posterior semicircular canal.

N.B.—Utricle detached.

upper surface of the petrous into the petro-squamosal sinus in infants. In adult life the subarcuate fossa becomes filled up by cancellous bone, and only survives as a small venous canal opening on the superior border of the petrous.

The posterior canal lies $\frac{1}{8}$ in. or less distant from the posterior surface of the petrous. Its arch encloses the opening of the aqueduct of the vestibule. In the infant its upper crus is visible upon the surface forming the posterior arcuate eminence, while its lower limb lies in the

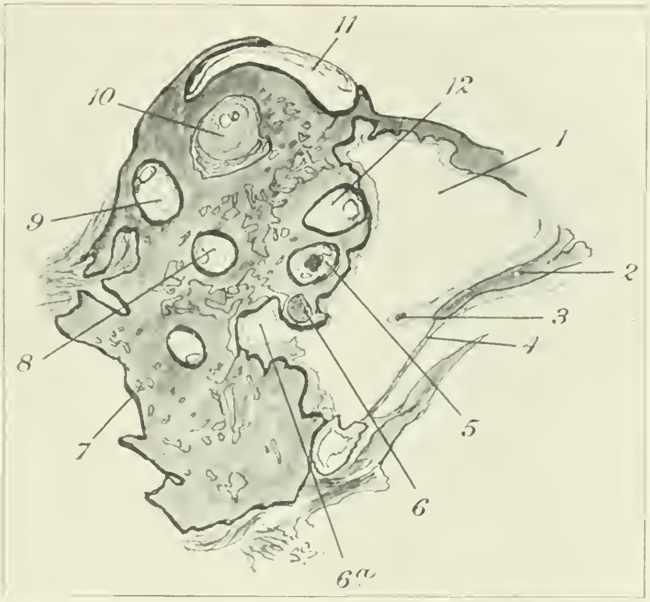

FIG. 4.

Vertical Coronal Section of Human Labyrinth, with semicircular canals.

(1) Attic.
(2) Outer attic wall.
(3) Chorda tympani.
(4) Tympanic membrane.
(5) Facial nerve.
(6) Stapedius muscle.
(6a) Sinus tympanicus.
(7) Inferior crus of posterior semicircular canal.
(8) Inner crus of external semicircular canal.
(9) Superior crus of posterior semicircular canal.
(10) Fossa subarcuata superior, with dura mater and blood-vessel.
(11) Superior semicircular canal, with artificial opening in roof.
(12) Outer crus of external semicircular canal.

roof of the jugular fossa and immediately internal and posterior to the deep sinus tympanicus. This relation with the roof of the jugular fossa is preserved in the adult when the fossa is deep.

The outer limb of the external canal lies exposed in the inner wall of the aditus, forming the external arcuate eminence; its ampullary end lies immediately above the facial nerve. This outer limb is exposed to damage in the removal of the "bridge" and posterior meatal wall in the performance of the radical operation. Between the superior and external canals lies a wedge-shaped mass of cancellous bone, which has replaced the fossa subarcuata. Caries not infrequently penetrates this area from the inner wall of the aditus, above the external canal.

The vestibule is lined by a thin fibrous layer, which encloses the perilymphatic space. The branches of the vestibular nerve in their course within the vestibule lie outside this fibrous layer and outside the perilymph, passing directly to the end organs in the membranous labyrinth, through the broad attachments which this has to the fibrous lining of the vestibule. The ampullary nerves are of surprising size, that for the joint supply of the superior and external ampullæ being comparable in transverse section to the facial itself. This nerve trunk is reconstituted, after being broken up in passing through the openings in the recessus ellipticus, and passes outwards and then backwards immediately outside the perilymphatic cavity and under shelter of, and slightly above the tympanic part of the facial nerve. The branch to the superior ampulla sweeps upwards, while that to the external continues its course backwards. The thin layer of bone lying between the facial nerve and this external ampullary nerve is compact, but has numerous narrow vascular lacunar spaces, a fact which may explain the symptoms of irritation of the nerve which occasionally occur where there is caries of the wall of the Fallopian aqueduct in its tympanic part. The nerve to the ampulla of the posterior canal enters the vestibule through a series of minute holes at the bottom of the short canal of the foramen singulare, is reconstituted, and passes as a short thick trunk to the posterior ampulla, lying upon the posterior and inmost portions of the floor of the vestibule.

The *fossula rotunda* is a deep depression passing upwards from the lower and posterior part of the tympanic cavity, under shelter of the free hooded margin of the postero-inferior part of the promontory which is marked by a corresponding deep upward notch. In the anterior wall of this fossa, and facing almost directly backwards with a slight downward inclination is the fenestra rotunda, closed by the membrana secundaria tympani. The sinus tympanicus may pass backwards and upwards for some distance internal to the Fallopian aqueduct, towards the concavity of the external semicircular canal, and in such cases is separated from the lowest part of the vestibule by an excessively thin layer of bone. When the jugular vault is very deep it may so far push up the floor of the tympanum as to seriously narrow or almost close the entrance to the fossula rotunda.

The *fenestra ovalis* lies at the bottom of the rather funnel-shaped pelvis ovalis. Its long axis passes backwards and downwards; its upper end is narrower than the lower. The upper wall of the pelvis is

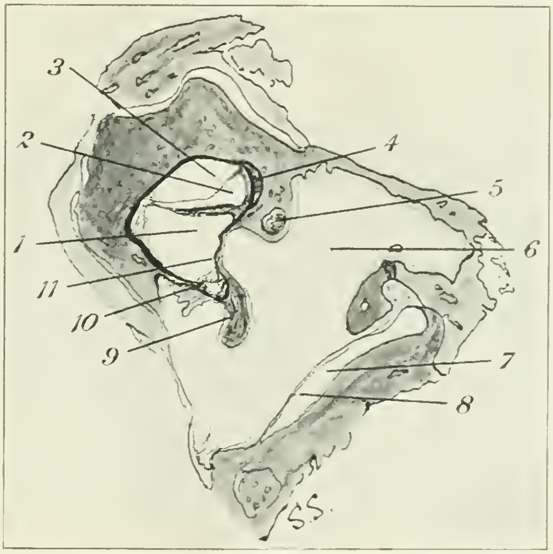

FIG. 5.

Vertical Coronal Section of Human Labyrinth, showing utricle, &c.

(1) Vestibule (perilymphatic space).
(2) Utricle and macula utriculi (lower wall).
(3) Artificial space, in preparation, upper wall of utricle becoming detached from wall of vestibule.
(4) Superior and external ampullary nerves.
(5) Facial nerve.
(6) Tympanic cavity.
(7) External auditory meatus.
(8) Tympanic membrane.
(9) Hooded margin of promontory.
(10) First turn of cochlea.
(11) Base of stapes.

deepened by the aqueduct of Fallopius, the lower by the promontory and the bar of bone which separates the pelvis ovalis from the fossula rotunda. The fenestra ovalis only admits of enlargement downwards.

The *cochlea* in outline forms a depressed cone. The centre of its base is formed by the anterior and lower part of the fundus of the internal auditory meatus. The axis round which it is wound points outwards and forwards and a little downwards. Its apex lies against the inner wall of the ostium tympanicum tubæ on a level with the musculo-tubal crest. The canal of the cochlea is spiral; it commences at the cochlear opening in the floor of the vestibule, makes a wide sweep of half a turn downwards, forwards and inwards, forming the eminence of the promontory, and, turning upwards, is then wound for two complete turns round the tapering central pillar or modiolus. In the posterior wall of the descending limb of the first half turn lies the fenestra rotunda. The junction of the ascending and horizontal parts of the carotid canal lies below the apex of the cochlea and a little in front of it. Only the first half turn is at all readily accessible from the tympanum. By the removal of the anterior wall of the bony meatus and opening up the first

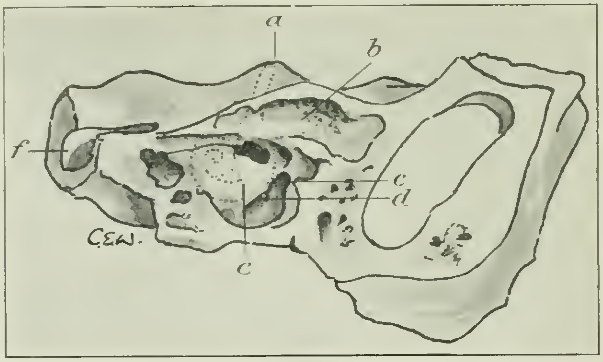

Fig. 6.

Vertical Section of Left Temporal Bone, showing the inner wall of tympanum and autrum, and their relation to the outer aspect of the labyrinthine capsule.

- (*a*) Superior arcuate eminence, containing inferior semicircular canal.
- (*b*) External arcuate eminence, containing external semicircular canal.
- (*c*) Sinus tympanicus: the latter lies on the opened sigmoid sulcus.
- (*d*) Opening of fossula rotunda.
- (*e*) Promontory containing first half-turn of cochlea.
- (*f*) Carotid canal.

half turn, the main body of the cochlea, lying in front of this and at a higher level, may be reached and destroyed. The base of the modiolus appears in the internal auditory meatus as the tractus spiralis foraminosus. The canals which convey the filaments of the cochlear nerve pass first axially and then turn outward into the lamina spiralis ossea, those situated externally in the tractus spiralis supplying the lower part of the cochlea, those centrally the apical part. Behind and above the main part of the cochlea, and directly in front of the upper part of the vestibule, lies the first part of the Fallopian aqueduct.

The course of the *facial nerve* lies at first within the cranium, passing outwards and immediately entering the internal auditory meatus, where it lies above the auditory nerve, the pars intermedia of Wrisberg intervening. The opening of the Fallopian aqueduct occupies the upper and anterior quadrant of the fundus of the internal meatus. Within the aqueduct the course of the nerve may be divided into three parts. The first is directed outwards and slightly forwards, and measures $\frac{1}{8}$ in. It ends in a somewhat expanded chamber lying behind the hiatus Fallopii

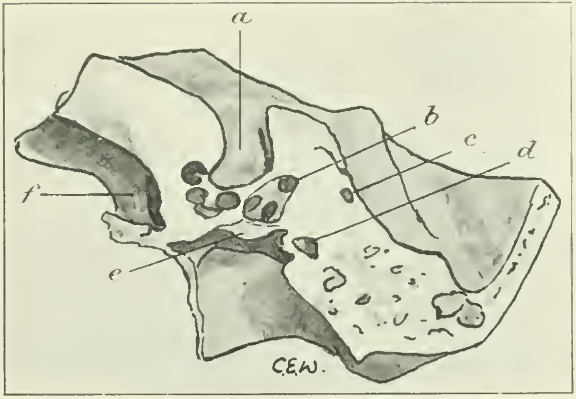

Fig. 7.

Horizontal Section of Left Petrous at level of lower border of fenestra ovalis, viewed from above.

(*a*) Internal auditory meatus: the canal of the foramen singulare has been opened up.
(*b*) Ampullary opening of posterior semicircular canal in the floor of the vestibule.
(*c*) Descending portion of posterior semicircular canal.
(*d*) Third part of aqueduct of Fallopius.
(*e*) Promontory.
(*f*) Opened carotid canal.

and housing the geniculate ganglion. The second part passes from this expansion backwards and downwards, at right angles with the first. Its course is straight and is inclined about 15″ to the horizontal; it measures nearly ½ in. Of this the central ¼ in. is visible in the tympanum, exposed on its outer and lower surfaces. The lower wall particularly is thin and often defective. This part of the canal has the fenestra ovalis below, the ampullary ends of the external and superior semicircular canals above, and the vestibule on its inner side. The last ⅛ in. of the second part is buried in the posterior tympanic wall, and lies immediately above and behind the little flask-shaped chamber of the stapedius muscle. The third part is about ½ in. long, passes downwards and a little outwards and backwards to the stylo-mastoid foramen, and is in relation with the deepest part of the posterior meatal wall. The angle between the second and third parts is about 120″.

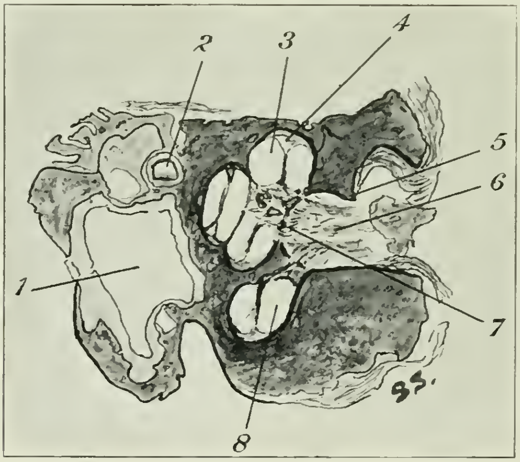

Fig. 8.

Vertical Coronal Section of Human Cochlea.

(1) Tympanic cavity.
(2) Mucous lined cell.
(3) Scala vestibuli of first turn of cochlea.
(4) Ductus cochleæ.
(5) Crista falsiformis.
(6) Auditory nerve entering fundus of internal auditory meatus.
(7) Base of modiolus.
(8) Scala tympani of first half turn of cochlea.

N.B.—Apex of cochlea not in this section.

The *internal auditory meatus* is lined completely by a sleeve of dura mater and arachnoid, whilst its lumen is occupied by a prolongation of the subarachnoid space, in which the seventh and eighth nerves lie. There is no definite continuation of this space along the seventh nerve in the aqueduct, but the nerve is surrounded by a quantity of loose connective tissue, while the dura mater becomes continuous with the lining membrane of the canal. The spaces of this areolar meshwork may thus be regarded as continuous with the subarachnoid space, though the meshes are close enough to prevent any free escape of cerebrospinal fluid if the sheath of the nerve is opened. The short canals through which the branches of the vestibular nerve pass into the labyrinth would seem to contain direct prolongations of the subarachnoid space, as when the nerves are avulsed in curetting the vestibule free escape of cerebrospinal fluid is the rule. Under such circumstances the subarachnoid space is virtually opened by the operation, and absolutely free drainage is essential to safety.

The aqueduct of the vestibule passes from its opening on the inner vestibular wall backwards in an arched course, which has its convexity upwards. The narrow canal ends by expanding into a flattened funnel-shaped chamber opening downwards and outwards under cover of a flat scale of bone on the posterior surface of the petrous. Above the upper margin of the opening lies the upper limb of the posterior semicircular canal.

III.—Morbid Anatomy and Pathology.

The surgical diseases of the labyrinth considered in this paper, and illustrated by the present series of cases, are those which originate as inflammatory processes in the osseous or membranous labyrinth, secondary to suppuration in the middle ear. The entrance of the infective agent was gained most often by destructive disease of certain definite and recognisable parts of the bony labyrinth, which we shall presently specify.

Infective Organisms.

The opportunities of examining the infective organisms of labyrinthitis have occurred in only a few cases. One of us cultivated pyogenic streptococci from the vestibule in a case of acute suppurative panlabyrinthitis, secondary to chronic suppurative otitis media (Case 27). In another similar case streptococci were obtained from the fluid in the internal auditory meatus post mortem (Case 2). Tuberculous disease of the petrous and mastoid process with secondary mixed pyogenic invasion of the semicircular canals was discovered in one case, and in another tuberculous disease had spread direct to the vestibule from the tympanic cavity (Cases 16 and 4).

Traumatism.

Traumatism played no important part in the causation of labyrinthitis in our cases. Once the external semicircular canal was opened unintentionally in the course of the radical mastoid operation by one of us, fortunately without any untoward result. In the case to which we refer the cortex of the mastoid was very dense and without cells. The identification of the opened canal posteriorly to the ampulla gave the first clue to the position of the antrum, which could not be recognised as a distinct cavity. The patient was watched with especial care for symptoms indicative of labyrinthine infection, but none arose.

In another traumatic case symptoms of mild irritation of the labyrinth were observed during the first twenty-four hours, with subsequent completely normal course. In a further case, which will be published elsewhere *in extenso*, on account of the post-mortem findings fatal acute panlabyrinthitis followed the radical operation. The autopsy showed that the oval window was filled by granulations, which had destroyed the stapes. Streptococcal invasion of the labyrinth resulted, although there was certainly no instrumental inoculation of the vestibule during the operation on the middle ear (Case 27).

Morbid Appearances and Course.

The morbid appearances and course of labyrinthine disease depend upon the extent and degree of the inflammation. Labyrinthitis may be (1) acute, or (2) chronic; either form may be (1) diffuse, or (2) circumscribed.

In *acute panlabyrinthitis* the membranous labyrinth becomes at first intensely injected, so that on opening the bony canal the membranous structures, instead of being invisible, appear as a brilliant opaque red filament. This condition we have seen in the external canal during operation (Case 10), as well as in the superior canal upon the post-mortem table (Case 27). We have not yet seen pus in the semicircular canals. The utricle and saccule are unrecognisable in the blood-stained and turbid fluid which fills the vestibule. Blood-stained and turbid fluid may be seen in the scalæ of the cochlea. This description of acute labyrinthitis is based upon data obtained from a complete examination of the labyrinth in one fatal case and upon the appearances observed during life in five other cases of acute labyrinthitis.

Histological Sections.

In sections of the cochlea, from a case of streptococcal labyrinthitis, fatal on the ninth day from known infection, we found round-cell infiltration and proliferation of cells in the ligamentum pectinatum and in the normally scanty connective tissue lining the scala vestibuli and scala tympani. The membrana basilaris, the organ of Corti, and Reissner's membrane were completely obscured by the inflammatory cells. The blood-vessels in the canals of the modiolus and in other parts of the cochlea were dilated and engorged.

The Spread of Infection.

The acute inflammatory process shows a marked tendency to spread along the perineural arachnoid sheath of the nerves in the internal auditory meatus. Sometimes there is a thick yellow plastic exudate matting together the nerves and the arachnoid sheath, in others the sheath is distended with only slightly turbid fluid; yet again in other cases the effusion surrounding the nerves may be virtually clear and show no deposit. These last cases form a distinct clinical group, to the consideration of which we hope to return upon another occasion. From the more plastic and opaque exudates organisms, notably streptococci, have been isolated, while at least in one case with only slightly cloudy effusion no organisms could be discovered in films or cultures.

The Labyrinthine Capsule.

The osseous, like the membranous labyrinth is involved in the acute form of labyrinthitis. The dense bony capsule of the labyrinth, when acutely inflamed, behaves as does compact bone in other situations of the body, and undergoes more or less extensive necrosis. Such necrosis may include virtually the whole of the cochlea, vestibule and semicircular canals. The lower turn of the cochlea may become an isolated necrotic fragment. The inner wall of the vestibule, the adjacent dense part of the wall of the internal auditory meatus, and the semicircular canals may undergo necrosis and separation in one composite mass.

It would seem that with free drainage from the labyrinthine cavity and the middle ear the acute inflammation may subside and the process terminate either in the formation of fibrous tissue in the labyrinth which may subsequently ossify, or in the sequestration of necrosed fragments with excessive production of granulation tissue and separation of the dead bone from the living bone in which it lies.

Chronic Infective Labyrinthitis.

Chronic labyrinthitis may be chronic from the beginning, spreading slowly from a local infective focus. It will in these cases be of a more or less circumscribed nature, but may at any stage give rise to an acute and diffuse process. On the other hand, chronic labyrinthitis may occur as the sequel of an acute labyrinthitis, when it will share in the diffuse character of the latter.

The area of infected bone is soft and crumbling, with granulations sprouting from the edges of fistulous tracks. We have on several occasions during operation observed a flattening and erosion of the promontory and of the external arcuate eminence. Where the canal is open its cavity may be plugged by a local growth of granulation tissue. In chronic cases affecting the canals or vestibule the cochlea is not necessarily involved, while if the lower turn of the cochlea is diseased the apical portion may still escape.

Character of the Middle Ear Disease.

In our experience labyrinthitis has been found far more frequently in chronic than in acute suppurative otitis media, yet there can be little doubt that in a number of cases the labyrinth was involved in the acute stage of the middle ear inflammation, which subsequently became chronic. This seems to account for those labyrinthine sequestra which are found with otitis media dating from early childhood, in which, after careful inquiry, no history of symptoms of labyrinthine disease could be obtained, while during the observed course of the case symptoms were likewise absent.

Path of Infection.

In the majority of cases at St. Bartholomew's Hospital the chronic disease in the middle ear was associated with perforation of some part of the outer labyrinthine wall. Certain regions of this wall appear to be especially vulnerable.

External Semicircular Canal.—A fistula of the external semicircular canal was found in fourteen cases.

Fenestra Ovalis.—A defect of the footplate of the stapes was found in six cases.

Promontory.—The promontory of the cochlea was perforated in five cases.

Fossula Rotunda.—In one case there was destructive caries and perforation of the lower part of the outer wall of the vestibule, through the fossula rotunda.

Site Unknown.—In three cases out of a total of twenty-six the site of infection was not discovered. We feel able to say that in these cases there was no perforation either of the promontory or of the external canal, but can make no statement with regard to the fenestra ovalis or the walls of the fossula rotunda.

Character of the Middle Ear Disease in relation to the Site of Invasion.—Cholesteatoma. Of the total of twenty-six cases eleven were associated with cholesteatoma. In six of these there was found a fistula of the external semicircular canal only, while in one a fistulous canal accompanied destruction of the footplate of the stapes and in two there was isolated destruction of the stapes. In the remaining two cases perforation of the promontory had taken place. Thus we see that cholesteatoma accounted for seven out of twelve of the cases of fistula of the external canal and for eleven out of twenty-six cases of infection of the labyrinth.

Tuberculous Disease.

Two cases of tuberculous disease occur in our series. In one perforation took place through the fenestra ovalis, in one through the external semicircular canal.

Acute Diffuse Labyrinthitis.

The presence of acute diffuse inflammation in the labyrinth was recognised in five cases, in three of which pus escaped when the vestibule was opened.

Chronic Labyrinthitis.

Granulations were removed from the vestibule in four cases of chronic labyrinthitis. Granulations were seen occluding the external semicircular canal in three cases in which there were no labyrinthine symptoms. In these cases the vestibule was not explored.

Sequestration.

Sequestration occurred in six cases. The cochlea was involved alone in two cases and in two others with the vestibule and semicircular canals. Necrosis of the vestibule and canals alone was found in two cases.

Fatal Cases: Cause of Death.

Death may ensue from meningitis or brain abscess or from other infective cause. Five cases terminated fatally. One succumbed to streptococcal septicæmia due to a virulent form of acute otitis media, complicated by acute labyrinthitis. In this case the labyrinth was not operated upon. Two died from coincidental cerebellar abscess (one not operated upon), one from consecutive lepto-meningitis, and one from acute internal hydrocephalus (labyrinth not operated upon).

Route of Intracranial Infection.—The path of the intracranial infection was found in three cases to be along the nerve sheaths in the internal auditory meatus, extending in one case to the basal meninges, in another to the cerebellum, and in a third direct to the fourth ventricle. The fourth case of intracranial lesion died from a cerebellar abscess which arose by direct extension through the dura mater over the posterior surface of the petrous.

Paths Recorded by other Observers.—Cases are recorded in which the superior canal alone has been eroded. This certainly seems to be possible in the light of our own experience, for it is not very rare to find infection of that area of the petrous which lies above the external semicircular canal and is occupied by the cancellous tissue intervening between the superior canal, the external canal, and the upper crus of the posterior canal. We can further believe it to be possible for erosion of the superior canal to be produced by an extradural abscess formed above the arcuate eminence. We have not met with this condition during life, though one of us has found defects in the superior arcuate eminence with a hiatus in the wall of the superior canal in macerated bones. As regards the abscess said to occur in the posterior cranial fossa by extension of disease of the labyrinth along the ductus endolymphaticus, we have so far met with no cases of this nature (Cf. Jobson Horne [13]).

IV.—SYMPTOMATOLOGY.

A careful analysis of the symptoms complained of and of those elicited by special tests in our cases shows that certain symptoms are common to considerable groups and have a definite bearing upon the diagnosis and treatment.

Group 1. *Total Absence of Labyrinthine Symptoms.*—Nine cases of manifest involvement of the labyrinth occur under this heading. Of these three presented a fistula or caries of the external canal; the fourth a defect of the stapes; the fifth was a case of accidental operative opening of the external canal behind the ampulla; in the sixth the first turn of the cochlea was opened by caries; in the seventh the cochlea was a loose sequestrum; in the eighth the oval window had been penetrated by tuberculous disease; while in the ninth the major part of the vestibule and canals was sequestrated.

Group 2. *Vertigo.*—Seeing that sixteen out of the total of twenty-six cases presented this symptom, we think it deserves somewhat detailed consideration. We define vertigo as the result in sensation of a discordance between the sense registers of any two of the paths of appreciation of space relationship. Clinically we apply the term to those cases in which there has been a definite direction of the sense of rotation, either of external objects or of the patient. In three cases, however, while the patient complained of the sensation of rotation, the planes of rotation appeared to be multiple, and could not be analysed; in a fourth case the plane of rotation was sagittal; in a fifth oblique. In the remaining eleven cases the plane of rotation was horizontal; in five of these we were able to make more detailed observations, and found that the patient experienced a sensation of movement of external objects from the affected to the sound side. In four of the cases there was a marked tendency to fall towards the affected side. Vertigo may be short lived, and disappear permanently after a few days. In other cases it may be prolonged over many months, either constantly present, recurrent with intervals of freedom, or of mild constant type with exacerbations.

Lesions Associated with Vertigo.—In eight cases the external semicircular canal was carious or perforated. In five cases there was a fistula into the vestibule. In one case we found caries of the cochlea to be accompanied by a fistula of the canal. In two cases the site of the lesion was not discovered. In no case was an isolated lesion of the cochlea associated with vertigo. While vertigo may obviously be regarded as a cardinal symptom of an affection of the vestibule or canals it is also clear that such a lesion may be present without any evidence of vertigo during the period of observation.

Group 3. *Vomiting.*—In fourteen cases vomiting occurred. In five of these we have no evidence of vertigo; all five were gravely ill, one unconscious; two others were suffering from cerebellar abscess and two from meningitis. In these cases it remains doubtful whether the vomiting can be directly ascribed to the labyrinthine lesion.

Group 4. *Tinnitus.*—In only three cases was tinnitus so obvious as to be made the subject of complaint. In all of these the lesion was vestibular in position.

Group 5. Deafness.—In two cases deafness was found to be absolute before operation. In all, hearing was defective. It is worth noting that in four cases in which the cochlea was involved in the operation hearing by bone conduction was more or less retained for C^1 fork. In two cases there was absolute loss of bone conduction.

Pain and Headache.—In four cases there was severe deep-seated pain in the ear. In four others there was severe headache of frontal, vertical, or occipital type, which was in each case immediately and completely relieved by the operation on the labyrinth. We have excluded cases of headache in which intracranial lesions were present.

Temperature and Pulse.—We have been unable to discover any common characteristics under this heading, although some elevation of temperature is the rule in acute labyrinthitis.

Spontaneous nystagmus appears to be a rare symptom, for we have not so far observed it.

V.—Operative Surgery and Records of Cases.

The two main conditions which demand surgical interference in the form of a planned operation on the labyrinth at present appear to us to be the incapacity produced by vertigo and danger of infective meningitis. Both of these indicate the posterior segment of the labyrinth as the primary point of surgical attack, for it is probable that in the great majority of cases it is by the short and open paths from the vestibule that infection travels into the subarachnoid space. In addition, the evidence is conclusive that the entrance of infection into the labyrinth takes place through some part of the outer wall of this segment in a preponderating number of cases, and the surgeon will thus be led by the track of infection to the same region. The outer wall of the vestibule is crossed by the facial nerve, which lies against its upper part. The lower part forms a portion of the inner tympanic wall, comprising the fenestra ovalis and the area below it down to the notch of the fossula rotunda. Above, and a little internal to the facial nerve lie the ampullæ of the superior and external canals, opening into the roof of the vestibule. We are thus presented with a choice of routes by which the vestibule may be opened: a superior, via the ampullæ; an inferior, through the tympanic wall. In all operations on the labyrinth it is essential that as much room as possible should be made available by the free removal of the boundaries of the preliminary radical operation and of the posterior meatal wall.

To open the vestibule from above, the external arcuate eminence is identified and removed, thus opening the outer limb of the external semicircular canal. This is now followed forwards above the facial aqueduct until the ampulla is opened. When this is effected the vestibular roof is completely removed by extending the operation forwards and upwards into the ampulla of the superior canal and the lower part of its ascending limb, and destroying the small pyramidal portion of very compact bone which lies between the proximal parts of the two canals. Such an operation we shall call "*superior vestibulotomy.*" It is a simple and short procedure, and the facial nerve runs little risk. It is, however, an inadequate and dangerous operation if carried no further. It is easily performed either with the burr or with cutting tools, but after a considerable experience with both we agree that a small and sharp gouge and a mallet give both the more expeditious and the safer opening. After opening the roof of the vestibule the external and superior ampullary nerves can be easily destroyed with a small sharp curette as they run on the anterior and outer wall at the level of the upper margin of the facial aqueduct. We have performed this operation in two cases, one of which was fatal from consecutive meningitis.

Case 1.—Male, aged 12. Cholesteatoma; erosion of external canal and roof of vestibule, left. Symptoms: none referable to vestibule; had recently had an attack of acute mastoiditis. Operation: radical; fistula of external canal leading into vestibule; this curetted but not opened below; no escape of cerebrospinal fluid. Result: meningitis on third day, death on fourteenth day.

Case 2.—Male, aged 15. Chronic suppurative otitis media, right; cholesteatoma; fistula of external canal; acute labyrinthitis; meningeal symptoms; cerebellar abscess. Symptoms: giddiness, vomiting, pain, headache, delirium, retraction; later abnormalities in reflexes. Operation 1: radical; superior vestibulotomy, exploration of temporo-sphenoidal lobe, drainage of cisterna pontis. Operation 2: exploration of cerebellum, re-exploration of temporo-sphenoidal lobe, drainage of lateral ventricle. Result: death with unopened cerebellar abscess.

Among Hinsberg's [10] cases of operation on the labyrinth were five of superior vestibulotomy (Cases 13, 17, 18, 19, 21), one with consecutive meningitis and death. Mr. Ballance [1] has published one case of superior vestibulotomy, with skin grafting of opening into the vestibule.

Superior vestibulotomy may be extended by the complete removal of the three semicircular canals. For this the burr is a practical necessity. To ensure complete removal the canals must be followed from end to end with a fine probe. After the preliminary opening of the external canal and ampulla, and the removal of the roof of the vestibule, the outer limb of the superior canal is followed over its convexity with a medium-sized burr until the crus commune of the superior and posterior canals is reached. In doing this there is considerable risk that the thin lamina of bone above the canal, on the upper surface of the petrous, will give way; and if the burr is being firmly used it may plunge through the dura mater and into the temporo-sphenoidal lobe. This has happened to one of us in three of the earlier operations, fortunately without any ill effects. The outer limb of the external canal is now returned to, and is followed backwards and then inwards with the same burr. As the deeper part of the convexity of the canal is reached the posterior canal will also be found to be opened in the posterior part of the deep bony cup which has been formed. The converging deeper part of the external canal and upper limb of the posterior canal can now be destroyed together as far as the vestibule, the crus commune being opened up. Finally the lower limb of the opened posterior canal is followed downwards, and then forwards and inwards to its ampulla and its opening in the floor of the vestibule. Vigilance must be exercised or the bulb of the jugular vein may be injured. It is well to take a smaller burr and follow carefully along the channel of the canal. We have only performed this operation once in a case of post-suppurative vertigo.

Case 3.—Female, aged 25. Post-suppurative vertigo, left ear. Symptoms: continued vertigo; exacerbations about every other day, in which patient falls; apparent rotation in vertical plane; has been sick with the attacks, but not recently; no nystagmus; pupils constantly dilated. Operation: radical; extirpation of canals; vestibule opened above but not below the nerve. Result: immediate intense vertigo, vomiting, nystagmus, and acute dementia; slight left facial paresis. Ultimate complete cure of symptoms, recovery of mental condition and face; left ear totally deaf to speech.

Four cases of superior vestibulotomy, with removal of the canals, have been published by Dr. Milligan [26]. Three of these were in non-suppurative cases. Mr. Lake [12] has published one case, also non-suppurative, in which superior vestibulotomy, with removal of the canals, was performed. At a later date the operation was extended to complete extirpation of the labyrinth.

The vestibule may also be opened below the facial nerve by the removal of its outer wall. This opening should be as complete as possible, and is attained by removing the stapes if this is present and cutting away the bone between the fenestra ovalis and the fossula rotunda. In this way all the accessible outer wall and a portion of the floor of the vestibule are removed, while it is not anatomically necessary that the membrana secundaria and the fenestra rotunda should be interfered with. No attempt must be made to gain room in a direction upwards from the fenestra ovalis. To do so is to risk a probable fracture of the bar of bone supporting the facial nerve or exposure of the nerve on its lower aspect, while the gain available averages less than 1 mm. The posterior ampullary nerve may be destroyed by introducing a curette and cutting against the inner and posterior part of the floor of the vestibule. We shall call this operation "*inferior vestibulotomy*." We have only one case of simple inferior vestibulotomy.

Case 4.—Male, aged 1. Tuberculous disease of tympanum invading vestibule, left. Symptoms: none of labyrinthine origin. Operation: radical; fistula leading into vestibule through tympanic wall; inferior vestibulotomy. Result: recovery with complete healing. Used for some time to fall over to left on being sat up in bed.

Dr. Milligan [21] has reported three cases of inferior vestibulotomy: one for post-suppurative fixation of the stapes, with grafting of the opening; two, during the course of suppuration. Hinsberg [11] has reported one case where this operation was done for diagnostic purposes.

Inferior vestibulotomy may be extended by the partial or complete removal of the cochlea. The first half turn is easily opened up and destroyed by working forward from the vestibular opening across the promontory. The remainder of the cochlea lies further forward, deeper and higher, and operative interference is much embarrassed by the anterior wall of the bony meatus. This should be removed down to the annulus tympanicus by cutting forceps, and the thick periosteum of the tympanic plate pushed forward and held aside by a narrow retractor. Additional room may be gained by removal of the floor of the meatus, and this is of particular value where the field of operation is contracted by a bulging cranial floor or by the prolapse of the dura mater of the middle fossa after a previous operation involving a large removal of the tegmen. By removal of both floor and anterior wall much comfort is obtained in cases of forward lateral sinus, the attack upon the labyrinth being made more directly against its outer wall. After removal of the anterior wall of the meatus the cochlear canal may now be followed from the open first half turn. When not softened by caries the body of the cochlea presents considerable resistance to attack. It should be opened on its outer and posterior aspect, when the modiolus can be broken up and destroyed by a burr or a stout small spoon. The lower and anterior part of the cochlear capsule should not be interfered with. The position of the knee of the internal carotid must be borne in mind lying below and in front and rather internally. So long as it is remembered it runs little risk of injury. To effect the destruction of the cochlear nerve this complete opening of the cochlea is necessary. For operations carried out below the facial aqueduct either a small gouge may be used with the mallet or a motor-driven burr; the latter is of great assistance in the total removal of the cochlea, but is apt to run rather wildly and endanger the facial nerve. We have now at St. Bartholomew's a motor which is capable of being reversed so as to drive the burr either clockwise or counter-clockwise; in this way the drag of the burr is directed away from the danger zone in all circumstances. We have performed *inferior vestibulotomy*, with partial or complete removal of the cochlea, in four cases.

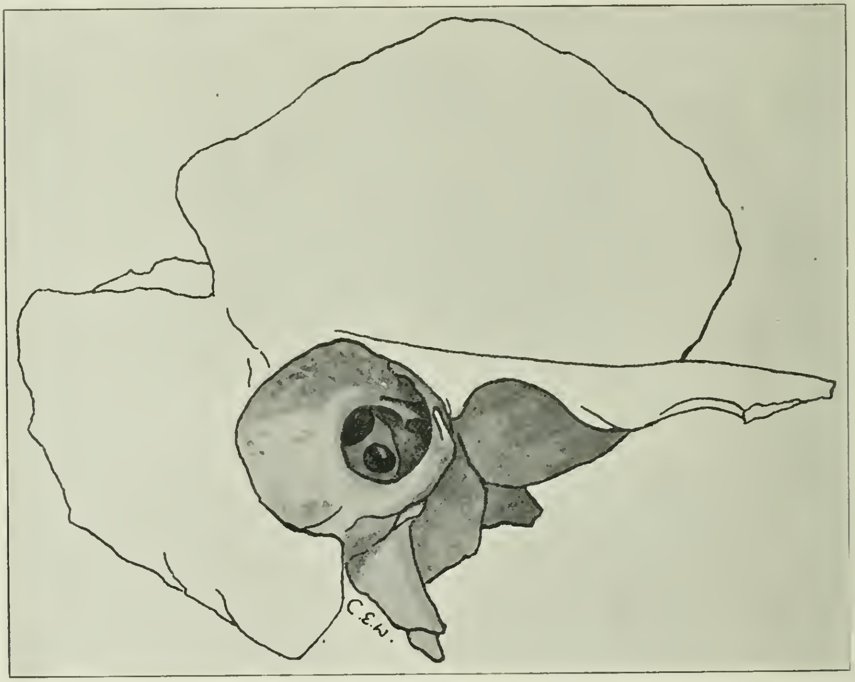

Fig. 9.
Inferior Vestibulotomy with partial removal of cochlea.

Case 5.—Male, aged 25. Chronic suppurative otitis media, right: defect of oval window. Symptoms: vertigo, vomiting, loud hammering tinnitus, total deafness. Operation: radical; inferior vestibulotomy and removal of first turn of cochlea. Result: vertigo five days, then ceased; slight facial paresis; still under treatment, February, 1908.

Case 6.—Male, aged 9. Chronic suppurative otitis media, left: cholesteatoma; fistula of promontory; chronic granulating labyrinthitis. Symptoms: none observed; condition discovered at operation. Operation: radical; inferior vestibulotomy, with opening of first turn of cochlea; cerebrospinal fluid escaped for forty-eight hours. Result: uninterrupted recovery.

Case 7.—Female, aged 20. Chronic suppurative otitis media, right: fistula of vestibule through fossula rotunda. Symptoms: constant headache and vertigo; objects moving from right to left; incoördinate walk, straying from left to right; falls suddenly and hurts herself; tinnitus; no vomiting; temperature 100° F. to 101° F., pulse c. 65. Operation: cavity of previous radical operation reopened; probe entered through fossula rotunda; vestibule and cochlea full of granulations; inferior vestibulotomy with complete removal of cochlea; escape of cerebrospinal fluid twenty-four hours. Result: immediate loss of vertigo and tinnitus; complete healing; temporary facial paresis [27]. (Patient shown to Royal Society of Medicine [Otological Section], December, 1907 [28]).

Case 8.—Female, aged 13. Chronic suppurative otitis media, left: cholesteatoma; destruction of promontory and first turn of cochlea. Symptoms: headache, vomiting, convulsions, unconsciousness. Operation: radical; inferior vestibulotomy with removal of cochlea. Result: complete recovery. After healing: Weber to left, Rinné, left negative; no perceptible loss of bone conduction; watch (repeated test with right ear closed and eyes shut): 7 in. in left ear; voice: quiet speech well heard when right ear closed by finger.

We have found *inferior vestibulotomy* with extension to the first turn of the cochlea a good and adequate operation, and are inclined to think it all that is necessary for those cases of infection of the vestibule in which there is no fistulous track leading into one of the canals. These two operations, superior and inferior vestibulotomy, and their respective extensions to the canals and cochlea, may be combined in various ways according to the indications. Of these combinations the most frequent and important is that which employs both superior and inferior vestibulotomy, constituting *double vestibulotomy*. While the methods we have

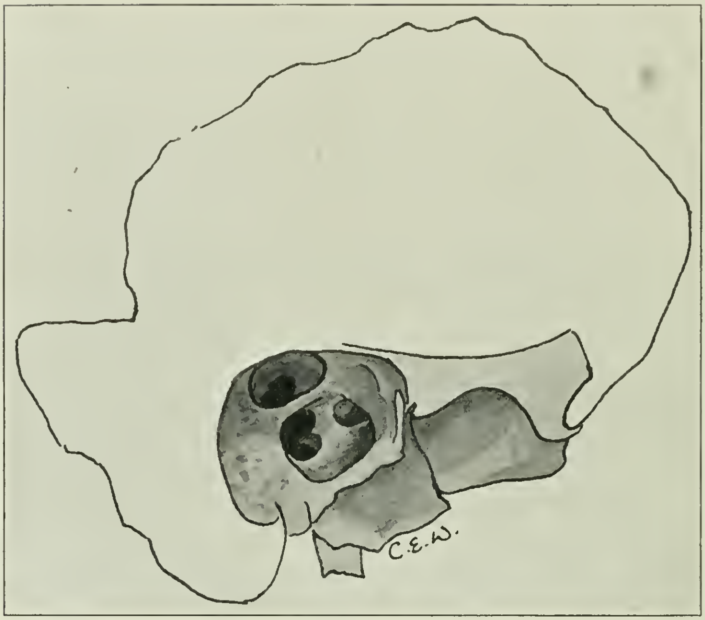

FIG. 10.

Double Vestibulotomy.

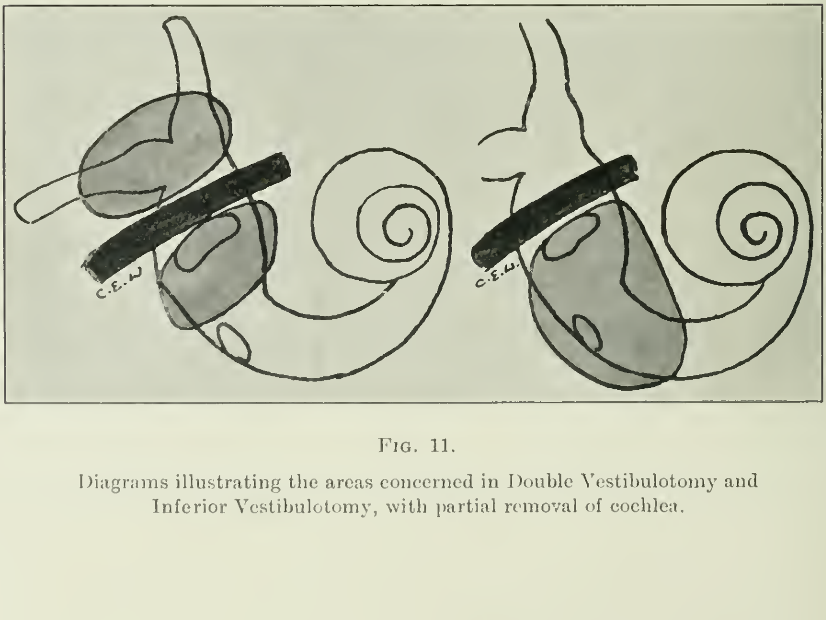

FIG. 11.

Diagrams illustrating the areas concerned in Double Vestibulotomy and Inferior Vestibulotomy, with partial removal of cochlea.

practised differ in some particulars, the operation is essentially that described by Dr. Milligan at the British Medical Association meeting, 1907, under the title of the "bridge operation." While putting forward no claims to priority, we may say we had independently evolved and carried out this operation, with extensions and total removal of the cochlea and canals, in April, 1904, in ignorance at that time of the pioneer work which had especially been carried out by Jansen [14] and Hinsberg [10], on which attention was first focused in this country by Milligan [20] and Whitehead [38]. The escape of cerebrospinal fluid, which is common where the vestibule is opened below the facial nerve, occurs either through injury to the inner wall of the vestibule or very frequently after quite light use of a curette within its cavity. When it occurs it is as a rule profuse, and lasts from twenty-four hours to a week, or even longer. It may give considerable trouble by the rapid filling of the cavity at the time of operation. We have performed the operation of *double vestibulotomy* without interference with the cochlea on four patients.

Case 9.—Male, aged 33. Chronic suppurative otitis media, left; cholesteatoma; caries of footplate of stapes. Symptoms: constant vertigo with paroxysms, accompanied by vomiting; objects appear to move from left to right; patient feels movement from right to left; vertigo increased by meatal compression; headache; pain in ear; sways to left; constant tinnitus; Weber referred to right. Operation: radical; probe entered fenestra ovalis without resistance; double vestibulotomy. Result: immediate disappearance of all symptoms; gradual development of facial paralysis with recovery. (Published *in extenso* [27].)

Case 10.—Male, aged 19. Chronic suppurative otitis media, left; acute labyrinthitis. Symptoms: giddiness, vomiting, pain; temperature 99·6° F., pulse 130 to 150. Operation: radical; exploration of external canal; double vestibulotomy; inflamed membranous canal. Result: disappearance of all symptoms; uninterrupted recovery; left ear absolutely deaf.

Case 11.—Female, aged 5½. Chronic suppurative otitis media, right; mastoid fistula, right; cholesteatoma; fistula of external canal; fistula of fenestra ovalis. Symptoms: right mastoid abscess in infancy; persistent fistula; recent horizontal vertigo, objects moving from right to left; at time of operation no special examination for vertigo, nystagmus, &c. Operation: radical; cholesteatoma; fistula of external canal; defect of footplate of stapes; double vestibulotomy. Result: complete healing; four years later—hearing (right): watch *nil*; Rinné negative. (Published *in extenso* [27].)

Case 12.—Female, aged 7. Chronic suppurative otitis media, left; cholesteatoma; defect of footplate of stapes. Symptoms: none referable to labyrinth. Operation: radical; discovery of open fenestra ovalis; double vestibulotomy. Still under treatment, February, 1908.

Dr. Milligan [23] has made reference to ten cases of severe labyrinthine suppuration upon which he has performed the "bridge operation." Hinsberg [10] includes one similar operation among his cases (Case 14). Mr. Secker Walker [31] has reported one case of double vestibulotomy.

We have three cases of *double vestibulotomy with partial removal of the cochlea.*

Case 13.—Male, aged 38. Chronic suppurative otitis media, left; cholesteatoma; fistula of external canal; acute suppurative labyrinthitis. Symptoms (ten weeks before operation): initial giddiness and repeated vomiting; vertigo, horizontal; objects moving from left to right; said he walked as if drunk. At time of operation: giddy only on movement of head; incoördinate swaying gait; no perception of sound on left side; no nystagmus; temperature and pulse natural. Operation: radical; external canal open; defect of roof of vestibule; double vestibulotomy, with partial removal of cochlea. Result: loss of all vertigo and of feeling of insecurity; complete recovery; healing. Facial paresis, which rapidly recovered.

Case 14.—Male, aged 13. Chronic suppurative otitis media, left; caries of external canal; acute labyrinthitis. Symptoms: sudden vertigo with vomiting; severe headache; seven days later giddiness and sickness have ceased; no nystagmus; continued severe headache. Operation 1: radical; surface of external canal eroded, wall softened; contents, dark-coloured fluid; vestibule contained pus; double vestibulotomy, with partial removal of cochlea. Result: relief of all symptoms, but incomplete healing of inner wall of the cavity; slowly developing facial paralysis; later, renewal of headache. Operation 2 (after eight weeks): removal of sequestra, consisting of inner wall of vestibule and inner ends of canals, and of cochlea by posterior and anterior routes. Result: complete healing; loss of headache; still under treatment (recent case).

Case 15.—Female, aged 46. Recurrent suppuration in cavity of old radical operation, right; caries of promontory. Symptoms: giddiness; pain; rapid loss of bone conduction. Operation: cavity reopened; curettage of granulations led into lower part of vestibule; double vestibulotomy, with partial removal of cochlea. Result: loss of vertigo; still under treatment.

Hinsberg [12] has reported three cases of double vestibulotomy with removal of the cochlea (Cases 15, 16, 20).

We have reserved the term "*extirpation of the labyrinth*" to describe the complete opening of every portion of the bony chamber, *i.e.*, double vestibulotomy with destruction of the whole length of each canal with the whole of the cochlea. We have three cases of this description.

Case 16.—Female, aged 11. Tuberculous disease of middle ear and petrous, left; fistula of external canal; chronic tuberculous labyrinthitis; acute infective labyrinthitis. Symptoms: severe frontal headache; frequent vomiting; pain in the head; temperature 100·8° F., pulse 140; no note of vertigo. Operation: radical; fistula of external canal; tuberculous abscesses in connection with posterior and superior canals; complete extirpation. Result: complete healing. (Published fully elsewhere [34].)

Case 17.—Female, aged 19. Chronic suppurative otitis media, left; site of lesion not discovered. Symptoms: vertigo, horizontal, from left to right; constant frequent falls. Operation 1: radical; cavity healed except region of external canal; vertigo persisted. Operation 2: extirpation. Result: slight giddiness and nystagmus for a few days, followed by a disappearance of symptoms; marked circus movements without giddiness on getting up, which gradually subsided; complete healing.

Case 18.—Female, aged 22. Chronic suppurative otitis media, right; necrosis of cochlea; caries of external canal, right. Symptoms: local pain; ataxic gait, drifts to right when walking, tends to fall to right when standing or sitting; no nystagmus; ocular movements and discs normal; pupils natural; knee-jerks normal; fine movements of right hand badly executed; right grip distinctly weak; slow pulse, 60; low temperature, 96·6° F. Operation: extirpation of labyrinth. Result: disappearance of symptoms. (Published fully elsewhere [34 and 35].)

Mr. Lake [17] has reported two cases of extirpation of the labyrinth for non-suppurative conditions, in one of which removal of the canals and vestibule, in the other [18] destruction of the cochlea, had been undertaken by previous operation. Harmon Smith [28A] has recorded one case of extirpation of the labyrinth for caries.

Where there is merely local caries of the external semicircular canal it has often been found sufficient to simply curette away all softened bone. Such a procedure will frequently result in the immediate relief of vertigo, showing that the lesion was an irritative one only. In these cases the ampulla is not opened, and in some the caries has been so superficial that the lumen of the canal has not been laid bare. These minor labyrinth cases have not been enumerated in our series. When curettage leads to the free opening of the ampullary part of the canal the result is virtually superior vestibulotomy and shares its risks if the vestibule is not afforded freer drainage than can be secured above the facial nerve.

We have carried out a *simple curettage* of the carious portion of the labyrinthine capsule in six cases, none of which have been subsequently submitted to further operation on the labyrinth.

Case 19.—Male, aged 39. Chronic suppurative otitis media; cholesteatoma; erosion of external semicircular canal, left. Symptoms: history of giddiness, but no evidence of vertigo lately. Operation: radical; opening of canal very wide, but ends plugged by granulations; nothing further done; recovery with some feeling of unsteadiness at first.

Case 20.—Male, aged 37. Caries of external canal, right. Symptoms: no giddiness; no nystagmus. Operation: radical; curettage of carious canal; drainage of extradural abscess in posterior cranial fossa; subsequent chronic osteo-myelitis of whole skull. About nine months afterwards died from frontal abscess, secondary to disease of frontal bone.

Case 21.—Male, aged 60. Chronic suppurative otitis media, left; cholesteatoma; erosion of external canal. Symptoms: no history of giddiness, although a private patient under close observation. Operation: radical; curettage of canal; no further symptoms developed.

Case 22.—Male, aged 14. Chronic suppurative otitis media, right; spontaneous facial palsy; fistula of external semicircular canal; fistula of Fallopian aqueduct. Symptoms: otorrhœa since aged 6, then mastoid abscess opened and healed. Three months ago giddiness lasting four days; fell once; no vomiting; six weeks ago facial palsy began. At time of operation no vertigo or incoördination. Operation: radical, external canal open; softened chocolate-coloured contents; outer part of facial canal open; bone soft; granulations at margins of fistulæ; curettage of canal; vestibule not opened. Result: no vertiginous symptoms; no change in facial palsy; otherwise recovered from middle ear suppuration.

Case 23.—Male, aged 59. Chronic suppurative otitis media, right antral cholesteatoma; fistula of external semicircular canal. Symptoms: otorrhœa and deafness, no vertigo. Operation: radical; antral cholesteatoma; fistula in ampulla of external canal curetted. Result (two days later): in erect position of head, eyes closed, falls back and to the right, sensation of objects moving up and to the left; head flexed and face averted downwards and towards the left, sensation of giddiness greater than in any other position of head; initial lateral nystagmus noted (direction of fixation of visual axes not stated); imperfect coördination of fingers. Three days after operation: nodding head (eyes closed) causes giddiness and falls forwards; says feeling as if falling backwards; horizontal shaking of head causes only slight dizziness.

Case 24.—Male, aged 42. Chronic suppurative otitis media, right. Symptoms: recent acute Bezold's mastoiditis, for which the radical operation had been performed; subsequently developed vertigo, horizontal type. Readmitted, second operation: caries of outer part of aqueduct; curettage of bone, exposing facial nerve; complete relief and recovery.

Hinsberg [10] mentions two cases of Körner's (Cases 1 and 2) of local curettage. Dr. Milligan [24] has published two cases of curettage of the external canal and Mr. Tilley [30] has reported one case.

Sequestra.—Sequestra of the labyrinth seem to involve either the vestibular system or the cochlea. While sequestra of both parts may occur in the same labyrinth we have not met with a case in which the whole labyrinth formed a continuous massive sequestrum. Sequestra of the cochlea are comparatively easily removed, as they have a broad surface towards the tympanum and are not enclosed by living masses of bone, nor is their extraction complicated by any dangerous relation to the facial nerve. The converse is the case with sequestra of the vestibule and canals. They lie deeply to the inner side of the facial spur and the posterior part of the base of the petrous. The direction in which they tend to sequestrate is towards the posterior surface of the petrous. In addition they must almost always involve the fundus of the internal auditory meatus, with its added risks. Sequestra of the vestibule and canals have to be looked for, and should be looked for, if there is serious ground for suspecting their presence. They may be discovered by probing through a fistulous track, either through the external canal or through the outer part of the vestibule, or may be betrayed by the persistent formation of granulation tissue in excess and refusal of the tissues to heal after the radical operation. It may be necessary to cut away apparently intact portions of the labyrinth before any part of the dead mass is exposed, for the typical sequestrum of the vestibule and canals does not extend to the outer surface of the labyrinth, and may be connected with it by a very narrow track of diseased tissue, along which infection has originally passed. Large sequestra may present considerable difficulty in extraction, as the available space is divided by the facial nerve into upper and posterior and lower and anterior sections. Removal may be effected by either of these routes, but in the majority of cases the upper is preferable as giving much greater scope for enlargement. The sequestrum must be freely exposed, and to do this a large removal of the posterior part of the petrous and of the wedge of bone between its upper and posterior surfaces may be necessary, after which the exposed dura mater can be held aside. This must be carefully done, for its outer surface will frequently be covered by granulations and its substance softened, the sequestrum having extended to the posterior aspect of the bone in the region of the posterior semicircular canal. We have three cases of *sequestrotomy*, in all of which the sequestrum was removed by the *posterior route*.

Case 25.—Male, aged 11. Chronic suppurative otitis media, left; facial palsy; fistula of external semicircular canal; granulations; no history of vertigo. Operation: opening up of petrous, radical; fistula of external semicircular canal, with granulations in canal; probing of fistula; detection of movable bony mass; opening up of petrous; removal of sequestrum, which consisted of vestibule and canals, and posterior wall of internal auditory meatus. Result: face improved subsequently; cavity completely healed. Sequestrum shown by Mr. West at the meeting of the Otological Society.[1]

Case 26.—Female, aged 3½. Chronic suppurative otitis media, right; facial palsy, right. Autopsy: cerebellar abscess. Symptoms: chronic suppurative otorrhœa; tender mastoid. Operation: complete post-aural; pus in antrum; one month later, wound not healed, sequestrum of petrous involving canals removed; two months later died of cerebellar abscess. Autopsy showed extensive caries of the petrous, involving labyrinth; nerves passing into right internal auditory meatus red and swollen.

(The above case was reported *in extenso*, with another intracranial case, in the *St. Bartholomew's Hospital Reports*, xxxix., by Mr. West.)

The third case of sequestrotomy has already been mentioned (*vide* Case 14) under double vestibulotomy with partial removal of cochlea. It illustrates the combination of *both routes*.

Mr. Fagge [6] has reported a case of removal of a sequestrum of the cochlea by the anterior route. Mr. Bull [4] has reported a case of removal of a sequestrum involving the vestibule and canals, with part of the internal auditory meatus, by the posterior route. Similar cases have been described by Mr. Hugh E. Jones [15], Mr. Fagge [7], and Mr. Cheatle [5]. Mr. Lake [19] reported and showed a case of total necrosis of the labyrinth removed by the combined routes.

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[1] *Trans. Otol. Soc.*, 1907, vol. viii.

Addenda.

To the above series of twenty-six cases illustrating the operative surgery of the labyrinth we append notes of four labyrinth cases which were not operated upon. Each case presents some clinical or pathological feature which has a bearing on the surgery of the labyrinth. The first case was one of acute streptococcal labyrinthitis, with symptoms developing after the radical operation on the mastoid and middle ear. Death was due to acute internal hydrocephalus, secondary to perineural arachnoiditis of the seventh and eighth nerves.

Case 27.—Male, aged 14. Chronic suppurative otitis media, left; subsequent suppurative panlabyrinthitis; death. Symptoms: long period of otorrhœa: no giddiness; blind from corneal ulcers. Operation 1: radical, hampered by large lateral sinus; granulations in tympanum and antrum; no ossicles. Result: vomited for next five days, then violent headache for three days. Operation 2: exploration of extradural area of previous operation cavity. Result: next day, *i.e.*, ninth day from infection of labyrinth, temporary relief of headache; sudden death, without antecedent loss of consciousness. Autopsy: defect of outer wall of vestibule, with granulations; no stapes; pus in vestibule; distension of arachnoid sheath in internal auditory canal about seventh and eighth nerves; acute ependymitis of choroid plexus of the fourth ventricle; acute internal hydrocephalus. Histology of the labyrinth: acute round-cell infiltration of the membranous labyrinth and lining of bony labyrinth, best demonstrated in cochlea (to be published fully elsewhere). Bacteriology: *Streptococcus pyogenes.*

In the next case the diagnosis rests upon clinical evidence only, as the labyrinth was not preserved for examination at the autopsy.

Case 28.—Male, aged 19. Acute otitis media, right (panotitis); streptococcal septicæmia. Symptoms: giddiness and vomiting on the fifth day of acute otitis media; rigors on seventh day; very giddy, could hardly stand; temperature, 104° F. Operation 1: radical; temporary relief of symptoms; renewed fever with delirium. Operation 2: exploration of brain negative. Result: death on ninth day of illness; no record of interior of labyrinth; no path of infection of labyrinth found; pure streptococcal cultures of heart's blood and spleen.

The third case which we give was one of spontaneous exfoliation of the cochlea, with perosseous sound conduction for C and C^1 forks apparently nearly perfect.

Case 29.—Male, aged 14. Chronic suppurative otitis media, left; necrosis of cochlea; sequestrum removed from meatus. Weber to left; Rinné negative left; perosseous conduction on left ear apparently normal after careful testing; watch heard 5 in. with eyes closed.

The last case was the one previously referred to as one of accidental opening of the normal external semicircular canal during the radical operation. No symptoms arose.

Case 30.—Male, aged 7. Chronic suppurative otitis media, bilateral; cholesteatoma in right tympanum; accidental opening of external canal. Operation: radical; dense mastoid; very narrow antrum; canal opened before cavity of antrum could be recognised. Result: no vertigo or subjective sensations could be found; no nystagmus; healing complete. Later: radical on opposite side; large mastoid cells; recovery.

VIa.—Special Tests.

We have found the following special tests of value in eliciting evidence of labyrinthine disease, and it has become our practice to apply them as a routine in all cases in which such a condition is suspected. We give the tests in the order in which their application seems most convenient:—

(1) *Rombergism.*—Standing with eyes closed, first on both feet together, then on each foot separately. We have found the rule to be that the patient sways or falls toward the affected side.

(2) *Gait, Walking Straight Forwards.*—An uncertainty of direction is usually manifest in cases of labyrinthitis where vertigo is present. The symptom reveals itself even with the eyes open. It may always be elicited when the eyes are closed. The tendency is to deviate toward the affected side, though this may be overcorrected by an obvious effort of readjustment. In the attempt to secure stability, the feet are placed wider apart than is natural, and in some cases, especially after destruction of the ampullary nerves, we have observed marked ataxic gait. In paroxysmal cases the attack is often so sudden and severe that the patient, if walking, falls as if in a fit and hurts himself.

(3) *Execution of Movements demanding precise Coördinate Control.*—For example, jumping or hopping forwards, backwards and sideways, with eyes (1) open, (2) closed. This test is inapplicable in some cases of severe vertigo. Other forms of coördinate movement may serve as a means of applying this test.

(4) *Elicited Nystagmus.*—It is only in our latter cases that we have made use of the turning stool, to the value of which our attention was drawn by Mr. Alexander Tweedie, of Nottingham. We have made use of rotation in a horizontal plane only. The patient is seated with the head erect and eyes closed upon an easily rotating chair. After ten revolutions movement is arrested and the patient is directed to open the eyes and fix them in extreme deviation, first to the side from which rotation has been made, then to the opposite side, the presence and the type of nystagmus elicited being noted in each case.

Normal Type of Nystagmus.

In the normal type the excursion is horizontal, and is characterised by a quick movement towards the side of deviation with a slow return.

Normal Reactions.—In normal states there is lateral horizontal nystagmus after rotation, upon deviation of the eyes towards the side from which rotation has been made, with absence of nystagmus on looking towards the direction of rotation. For instance, after rotation to the right, nystagmus is present when the eyes are directed to the left, and absent when the eyes are turned to the right.

Abnormal Reactions.—In some cases of unilateral lesion we have found it impossible to elicit nystagmus after rotation toward the affected side, while in others although nystagmus was produced it was not of the normal type.

Significance.—While we regard it as probable that rotation and similar tests will prove of extreme value as affording definite objective signs in cases of suspected labyrinthine lesions, we are unable at present to do more than accept an abnormal response as presumptive evidence of a disordered labyrinth.

VIb.—Indications for Operation on the Labyrinth.

Briefly, *the* indication for operation on the labyrinth is the diagnosis of infective labyrinthitis. This may be made (1) from the presence of symptoms and response to special tests; (2) from operative discoveries.

(1) *Symptomatic Indications.*—As we have intimated, vertigo is the most frequent of the cardinal symptoms. It is, of course, none the less true that vertigo, as an isolated phenomenon, is not diagnostic of labyrinthine disease. Again, many cases of labyrinthine disease have no vertigo at the time of operation. In some of these cases a definite history may be obtained, and we regard this as being almost as valuable

as the presence of the symptom. In sixteen of our twenty-six operation cases vertigo was known to have occurred at some period. The value of this symptom is greatly reinforced by association with one or more of the following: (*a*) vomiting; (*b*) loud tinnitus; (*c*) advanced or progressive loss of bone conduction; (*d*) local pain or headache. Many cases of chronic vertigo are at the same time markedly neurotic, and it may be by no means easy to estimate the value of their symptoms. It is in such cases that the results of the special tests become of great importance. In all cases they should be applied to confirm the patient's account of his sensations.

(2) *Operative Discoveries.*—Many obvious labyrinthine lesions are discovered accidentally during the course of the radical mastoid operation. A careful examination of the whole of the exposed area should in all cases be made, and will frequently reveal extensive labyrinthine disease. We consider that all cases in which the vestibule or the ampullary ends of the canals are involved should for the safety of the patient be thoroughly opened up and drained. In cases of fistula or caries of the outer limb of the external canal behind the ampulla presenting no labyrinthine symptoms we think the operator may be for the time content to merely curette away the softened area of bone. It is well to bear in mind that a fistula of the canal may also be a fistula of a sequestration cavity, which would be reached by a probe passed directly inwards. When the promontory of the cochlea is found perforated the diseased bone must, of course, be completely removed. In the majority of cases the operation will be led in this way into the vestibule. In all cases of operation of the promontory we think it is essential that free and adequate drainage of the vestibule should be established and maintained.

VII.—Results.

In every case in which *vertigo* was present at the time of the operation the patient has been completely relieved—in some immediately, in others after a short period, in no case exceeding two or three weeks. In no case was useful *hearing* preserved on the side of operation on the labyrinth; in the majority hearing was already absent or very defective before operation.

We have been fortunate in having no case of permanent *facial paralysis.* While there has been a *mortality* of 17 per cent., in only 3·3 per cent. (the one case of superior vestibulotomy) could the operation be considered to have led to this result. There have been no fatalities among the cases of inferior or double vestibulotomy.

Of the five fatal cases two were submitted to no operation on the labyrinth, and our opinion is that a timely operation could have saved the life of one of these.

We should like to conclude by expressing our great feeling of indebtedness to Mr. Cumberbatch for the encouragement and criticism that he has given to work which has often been anxious and tentative, for the support and advice without which our earlier cases would not have been attempted, and for his generous permission to make use of the clinical material at St. Bartholomew's Hospital.

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INDEX OF MORBID ANATOMY.

Necrosis of cochlea—18, 29.
Necrosis of vestibule and canals—25.
Necrosis of canals—26.
Necrosis of cochlea, vestibule, and canals—14.
Fistula of external canal—1, 2, 11, 13, 14, 16, 18, 19, 20, 21, 22, 23, 24, 25, 30.
Fistula of erosion of promontory—6, 8, 15, 18, 25, 29.
Defect of stapes—4, 5, 9, 11, 12, 27.
Fistula of fossula rotunda—7.
Not found—3, 10, 17.
Tuberculosis—4, 16.
Cholesteatoma—1, 2, 6, 8, 9, 11, 13, 19, 21, 23, 25.
Acute labyrinthitis—2, 10, 13, 14, 16, 27, 28.
Circumscribed granulations in external canal—19, 22, 23.
Circumscribed granulations in vestibule—6, 7, 15, 25.
Pus in vestibule—13, 14, 27.
Dark fluid in canal—14.
Red membranous canal—10, 27.
Extradural abscess—20 (? others).
Meningitis—1, 27.
Open Fallopian aqueduct, caries—9, 22, 24.
Accidental opening of external canal—30.
Post-suppurative—3.
Cerebellar abscess—2, 26.
Perineural arachnoiditis—1, 12, 26, 27.

INDEX OF SYMPTOMS.

Absent—1, 4, 6, 12, 20, 21, 23, 25, 29, 30.
Vertigo—2, 3, 5, 7, 9, 10, 11, 13, 14, 15, 17, 18, 19, 22, 23; after operation only—24, 28.
Drifts to right—5, 7, 9, 18.
Objects move to opposite side—7, 9, 11, 13 (? 17).
Objects move to same side as lesion—5 (? 17).
Gait tested in 3, 5, 7, 9, 11, 13, 17, 18, and in all defective.
Vomiting—1, 2, 3, 5, 7, 8, 9, 10, 13, 14, 16, 26, 27, 28.
Circus movements, after operation—17.
Vertical movements—3.
Oblique movements after operation—23.
Spontaneous facial palsy, before operation—22, 25.
Tinnitus—5, 7, 9, 10.
Deafness, absolute—3, 5, 7, 10, 13, 17, 18.
Deafness, partial—8, 9, 11, 12, 15, 16, 29.
Bone conduction tests, peross. nearly intact, apparently—8, 11, 12.
Watch—16, 8, 29.
Pain—2, 8, 9, 10, 15, 18, 25.
Headache—2, 7, 8, 9, 14, 16.
Meningeal symptoms, recovering immediately after operation on labyrinth—2, 8.
Sex—males, 19, viz.: 1, 2, 4, 5, 6, 9, 10, 13, 14, 20, 21, 22, 23, 24, 25, 27, 28, 29, 30.
Sex—females, 11, viz.: 3, 7, 8, 11, 12, 15, 16, 17, 18, 19, 26.
Age—under 5, two cases; between 5 and 10, four cases; between 10 and 20, twelve cases: between 20 and 30, four cases; over 30, eight cases.
Side of lesion—right, thirteen cases; left, seventeen cases.

DISCUSSION.

Dr. W. MILLIGAN congratulated the authors on a remarkably good piece of work. The paper was an exceedingly good résumé of the subject, and the compilation meant a vast amount of work. It was a credit not only to themselves, but to the aural department of St. Bartholomew's Hospital. As the authors mentioned, the subject of labyrinthine suppuration and its operative treatment was as yet comparatively in its infancy, and their classification of operations into superior and inferior vestibulotomy would be of great practical value. The whole question had interested him for a long time, and he regarded the paper as most instructive. A matter of practical and clinical interest was the comparative rarity of labyrinthine suppuration in acute ear cases. His own experience of labyrinthine involvement following acute suppurative middle ear disease was limited to two cases. One was in a little boy who had scarlet fever, with double acute mastoid infection, and on one side infection of the labyrinth through the external semicircular canal; the other case was very similar, but followed measles. With regard to cholesteatoma, he agreed with the authors' observations that it was one of the most common pathological conditions which was present and likely to invade the labyrinth. The presence of cholesteatomatous debris in the discharge should be regarded not only as dangerous, but the fact should be remembered that many cases of labyrinthine disease were the result of cholesteatoma. He agreed that superior vestibulotomy was a more or less dangerous operation, and that if possible the labyrinth should be approached by the inferior route, and that one need not hesitate to work comparatively far backwards in the region of the posterior semicircular canal. He asked what number of cases of labyrinthine suppuration the authors had met with in their ordinary work. His own investigation led him to the conclusion that about 1 per cent. of chronic suppurative ear cases presented labyrinthine symptoms. He did not know of any other observations on the point. There was considerable room for difference of opinion as to the methods of operating. The authors recommended the use of a fine chisel. He had had no experience of that as he had used the burr, using the gouge and chisel for the ordinary mastoid operations. The burr he used was longer than the ordinary one, and gave a little more room without diminishing the tactile sensation of the operator. It was interesting to know that the labyrinth could be opened by means of a mallet and chisel. Still, he thought that there was some risk of the chisel slipping. He insisted upon the best possible illumination, and the frequent use of adrenalin, which acted powerfully in diminishing hæmorrhage. With regard to the paths of infection, the statistics of the authors agreed very well with those of other authorities, viz., that the external semicircular canal was the main point of entry. There were a fair number of cases in which there was erosion of the external semicircular canal, cases which might be regarded as extra-labyrinthine. The bone was eroded, but there was no definite extension of inflammation to the interior of the labyrinth. It was a localized process, and probably very insidious, so that there was time for adventitious inflammatory products to be thrown out so as to localize it. At the operation it was a nice point whether it was advisable to continue the operation or whether it was not sufficient to gently scrape or burr the bone, and not to open into the labyrinthine cavity itself. Many pathological processes, even in the labyrinth, were purely localized, so that the posterior end of the internal ear might be infected and the inflammation localize itself there, the cochlea remaining healthy. It must be recognized that the presence of pus in the internal ear was a menace to life.

Dr. BRONNER congratulated the authors on their most interesting paper, which he regarded as the most valuable which had been brought before the Section. He asked what proportion of cases of suppuration of the labyrinth showed no severe symptoms. In regard to the point raised by Dr. Milligan as to whether scraping was enough in some cases, he (Dr. Bronner) had never done the classical operation, but in five or six he had simply scraped the diseased canal and the cases had healed up. The operation set out by the authors was not only very difficult, but dangerous, so that when only the canal was diseased and there were no definite symptoms of affection of the whole of the labyrinth, he should prefer to scrape the canal first and then, if necessary, perform the more serious operation.

Mr. WAGGETT heartily congratulated the authors on their valuable work. He drew attention to the paper by Richards in the *Laryngoscope*, and particularly to the point which that writer made concerning the modiolus, of which only the apical third could be removed without opening the internal auditory meatus. A neglect of this anatomical point had led in one instance to a fatal issue by meningitis.

Mr. YEARSLEY commented on the fact that only two out of the thirty cases were tuberculous; that was noteworthy in comparison with what Dr. Milligan and Mr. Whitehead had stated as to the rarity of tuberculous lesions in the labyrinth. In one case mentioned, the external semicircular canal had been opened and the antrum could not be recognised as a cavity. This was interesting after the very dogmatic statement which had been made that absence of the antrum never occurred. He recently had under his care a case of suppurative meningitis in which the labyrinthine lesions were not recognised until after death, and in which the path of infection to the meninges was through an erosion in the superior canal. That case developed spontaneous nystagmus.

Dr. FITZGERALD POWELL desired to record his thanks for this most excellent paper. He wished to know what the authors considered the special indications necessitating the opening up of the labyrinth, and what symptoms or definite points could be looked upon as indicating the presence of pus in the semicircular canals and cochlea.

Dr. DUNDAS GRANT asked whether the authors attached importance to the routine testing for the highest pitched tones of Galton's whistle. He had tested that in looking out for the possibility of labyrinthine trouble in some of his suppurative cases, and some writers, especially Ostmann, had pointed it out as an early sign worth noting.

Dr. ALBERT GRAY said the question of operating must be one of difficulty because the cases which died were known, while there must be many cases living who yet had labyrinthine trouble. The specimen from the case which he showed in the adjoining room had a suppurating ear when aged 60, and was absolutely deaf in the affected ear. Probably it had been affected all his life, and he went through life without a symptom until ripe old age and died from malignant disease elsewhere. Therefore it was an open question as to when operation should be done and when not. The danger of the operator destroying the modiolus of the cochlea had been mentioned. The upper part could be destroyed with impunity, but destruction of the lower portion might be dangerous, since in this case the cranial cavity was opened. If it was so easy to infect the arachnoid space one would not perform such a severe operation as that recommended in the *Laryngoscope* by Richards.

Mr. HUNTER TOD congratulated the authors heartily, not only on their paper, but on their having been able to collect so many cases. He noticed that the paper only dealt with suppurative lesions, as the opening of the semicircular canals for vertigo in non-suppurative cases was not discussed. The cases might be divided into three groups—tuberculous suppuration, acute suppuration and chronic suppuration. All had seen tuberculous disease of the mastoid involving the labyrinth, and in these cases it was usually sufficient to curette out gently the vestibule or the cochlea. Frequently facial paralysis already existed. In such cases the technique of the operation was rendered more easy owing to the fact that irreparable damage to the facial nerve had already taken place as a result of the disease. Many of such cases did very well. Mr. Tod's experience of acute suppuration of the labyrinth was limited to that occurring in children, and in which death occurred as a result of meningitis. With regard to chronic suppuration of the labyrinth, associated with chronic mastoid disease, Mr. Tod said he could not help being surprised that the authors had been able to collect together so many cases. Excluding the simple Schwartze's operation, he had performed at least 350 operations on the mastoid process during the last seven years. This operation had only been done when all other means had failed to cure, that is, after the usual conservative treatment, after frequent removal of polypi, and in many cases where ossiculectomy had also been performed. Although, whilst performing these operations he had always made a point of inspecting the inner wall of the tympanic cavity and the region of the semicircular canals, especially in cases in which there had been vertigo and internal ear deafness, yet with the exception of superficial caries of the external semicircular canal, which was not infrequently met with, he only remembered three cases in which there was evidence of any sign of disease within the labyrinth. In these cases he had contented himself with curetting away the area of granulations and had never felt justified in doing anything further. He quite admitted that he might have missed cases of labyrinthine inflammation, but the consequences did not seem disastrous. He could merely say that in all cases of uncomplicated mastoid disease on which he had performed the complete operation he had never had a fatal result. He admitted that in those cases in which healing after the operation was not satisfactory the continuance of the suppuration was almost invariably due to bone disease about the region of the promontory and floor of the tympanic cavity; but even in these cases healing, as a rule, eventually took place as a result of treatment. Although he considered the paper an admirable one, he thought, however, that the danger might arise of surgeons tending to operate too frequently on the labyrinth. From his own experience he would say the less that was done the better, and that operation on the labyrinth should be performed only when there were very definite symptoms of a suppurative lesion being present.

Mr. R. LAKE added his tribute of praise to the authors. Though he had been operating for a number of years, it was only comparatively recently he had started to operate on the labyrinth; yet previously all one's cases got well. In 1893 he reported two cases of external semicircular canal disease, neither of which was operated on, yet both got well. Operation often was rather a question of advisability than of necessity, and the former was the outcome of a fairly long experience. He would not say that the operations set out in the paper were difficult, but the authors seemed to have overlooked the posterior method of entering the vestibule, which was now coming into vogue in France; he meant entering posterior to the facial nerve. He asked whether there was any particular reason for removing the lower turn of the cochlea where the cochlea was not diseased. He had himself taken away the external wall of the vestibule several times, but he had not gone into the cochlea and could not see any reason for doing so.

Mr. FAGGE said that seven years ago, while in Germany, his attention had been drawn to labyrinthine suppuration, and ever since then he had been on the look-out for cases at Guy's. With the exception of some dozen cases of localized caries of the external semicircular canal, which only needed curettage, he could remember only two cases of suppuration in the labyrinth. In both of them he performed what the authors called superior vestibulotomy with a satisfactory result. His own experience agreed with Mr. Tod's; he could not believe that he had missed a large proportion of labyrinthine suppurations, because in none of the cases in which otorrhœa had continued after his complete mastoid operations had he noticed any symptoms of vertigo, tinnitus, or internal ear deafness, and none of these cases had died.

Dr. KELSON congratulated the authors on their paper, remarking that such contributions would do much to support the vitality of the Section. He asked whether their investigations had thrown any light on the physiology of these parts. There were cases in which the semicircular canals were dealt with, and others in which the cochlea was treated, and he asked whether any difference had been noted in irritative as opposed to destructive lesions. Those who thought of doing work of the kind were met by the remark that "in no case was useful hearing preserved." But he would like to hear what their idea of useful hearing was, as in another part of the paper it was stated that "the watch was heard at 7 in."

Dr. PATERSON asked as to the value of caloric nystagmus in the cases. It was an important question to distinguish between circumscribed disease, which may be left alone, and diffuse labyrinthitis, where there was very grave danger. The authors stated that in fistula where there were no symptoms one should be content to wait, and he would like to know to what extent they had adopted that course. He himself had cases of fistula in the external canal with labyrinthine symptoms, which had entirely cleared away, vertigo and nystagmus disappearing after the radical operation. Would it not be better to lay down a rule that the operator should wait a little, except where there were grave symptoms, and in them operate promptly? In the two cases of inferior operation he did he used a chisel, which he thought was more under control than a burr would be.

The PRESIDENT (Dr. McBride) joined in the congratulations which had been so well expressed; the paper would, no doubt, cast lustre on the Section. At one time he worked a good deal at the subject of vertigo. The authors had found, in five or six cases out of eleven, where the observation could be made, that surrounding objects seemed to move from the affected towards the sound side. That form of investigation might be followed further. Was there any difference in that according to whether the lesion was irritative or paralysing? In regard to Case 28, it was said that there was exploration of the brain; did they mean lateral sinus exploration? The question of nystagmus had been very largely studied by the Viennese school, which stated that labyrinthine nystagmus was increased on looking towards the sound side, whereas cerebellar nystagmus was increased on looking towards the diseased side. He asked whether the authors had inquired into that or whether they had cases on hand which would enable them to confirm or deny that. Another interesting point was heat nystagmus. That had scarcely been touched upon in English literature, but in Vienna it was looked upon as important. Barany laid down the rule that in the ordinary labyrinth, if one syringed the ear, whether the labyrinth was exposed or not, if the water were at the body temperature there was no nystagmus, if it were cold water there was nystagmus away from the side syringed, while if the water was hotter than the body there was nystagmus to the opposite side. It was supported by others, and he mentioned it because it seemed to be based on false reasoning. Zeroni (if he remembered right) wrote an elaborate paper on post-operative meningitis, and pointed out that in cases of radical mastoid, where the labyrinth was involved and there were granulations on the inner wall of the tympanum, it was dangerous to scrape such granulations, and he mentioned several deaths so caused.

Dr. DONELAN had noticed the absence from the list of authors referred to an important contribution to the 1906 and 1907 numbers of the Italian *Archivii di Otologia*, by Professor Gradenigo, entitled "Sulle Funzioni del Labirinto Non-Acustico" (The Functions of the Non-Acoustic Labyrinth). It dealt especially with nystagmus and with most of the other points referred to by the President.

Mr. WEST, in replying, said he wished to make a few remarks in supplement to the paper. He held strongly that removal of the whole of the canals was probably never necessary except in tuberculous cases in which the disease had extended along the canals. The ampullary nerves could easily be reached and destroyed in the vestibule, and after their destruction vertigo ceased. The complete destruction of the canals was an operation of very great difficulty and length, though it was possible, as in Case 18, which had indeed been carried out by the light of an ordinary oil lamp. When he did that operation, the earliest of the series, he had not learned how much or how little was necessary. He held that after the radical operation, in cases with continued vomiting, elevation of temperature, and especially severe vertigo, vestibulotomy was urgently called for as the patient was in grave danger of meningitis. If no relief was obtained in twenty-four hours, the internal meatus should be opened through the inner vestibular wall and drained. He had recently done this with success. Cases 1 and 27 had died from want of such drainage. In answer to the questions as to the statistics, they had had thirty-two cases of involvement of the labyrinth among 320 major operations conducted since the earliest of the series of cases, excluding minor cases of fistulous canal without symptoms and superficial erosion. This gave an operation frequency of 10 per cent. As regards case frequency in suppurative otitis media he could not give definite figures; but there had been about 4,500 out-patients during the period, and assuming that 60 per cent. of these were suppurative, this would give a figure, which was about the same as Dr. Milligan's, of 1 per cent. In regard to burr *versus* chisel, it was a matter of use and habit, but when working along a canal with a burr it was likely to get choked up with debris, while with a chisel a perfectly clear dissection could be carried out. They invariably used a head lamp in their operations. The cases of superficial erosion were not included in the totals. He did not think the results pointed to the operation being a dangerous one. In only one of the cases could death be ascribed to the labyrinthine operation, and that was an inadequate one; nor had they then recognized the possibility of drainage through the internal auditory meatus. In all cases where the facial nerve was not mentioned the face was not affected. Even in some cases where that nerve formed a bridge across a chasm there had been no symptoms; but if there was firm plugging on to the exposed nerve, then symptoms of facial paralysis occurred. The escape of cerebrospinal fluid had been so frequent in their cases that they had not regarded it with dismay, and with free drainage the risk of meningitis was very small. Of the three cases of pus in the labyrinth one at least was violently ill, with raised temperature, vertigo, vomiting and headache, and intense deep-seated pain in the ear. That series of symptoms pointed to suppuration in the labyrinth. They had not very completely investigated the tuning-fork reactions. In one case there was caries of the lower part of the promontory, and the patient had increasing loss of bone conduction. The advisability of operating in those cases rested on similar grounds to that of the ordinary radical mastoid operation. Extirpation of the cochlea was not advised, unless for tubercular disease or cholesteatoma. Otherwise, the opening of the first half turn was adequate. In no case where the cochlea was incompletely removed had there been tinnitus afterwards; that was a rare symptom of labyrinthine disease. They had had so many cases because they had had their eyes keenly open for them in the last three years, and men who knew they were working at the matter sent them cases. He congratulated Mr. Tod and Mr. Fagge on the uniform success of their mastoid operations, but he thought everyone had met with cases in which that result was not attained. He desired to draw Dr. Paterson's attention to the risk of not operating on the labyrinth when there were clear indications in the morbid anatomy for doing so. In answer to the President, there were no sensations, either subjective or objective, of rotation in paralytic lesions. Where patients had had violent vertigo before the operation, and after it, where there was destruction of the whole of the ampullæ or the vestibule, vertigo ceased. But if the labyrinth had been functional beforehand, there were ablation phenomena afterwards, the patient having sometimes an ataxic gait, and having a tendency to stray towards the operated side. Those symptoms disappeared after two or three months, but were apt to recur if the patient got anæmic or otherwise out of health. In Case 28 the lateral sinus was examined post mortem and was normal: it contained no clot.

Mr. TOD desired to say that he was afraid Mr. West had misunderstood him. He had not said that he invariably got an immediately successful result as a result of the complete mastoid operation. The point he wanted to emphasize was that in uncomplicated mastoid disease with no intracranial symptoms he had never had a death as the result of the operation. He was, however, quite prepared to admit the possibility of caries of the promontory or chronic suppuration of the labyrinth being a rare cause of failure of the complete mastoid operation to cure the suppuration. The other point he wanted to make was this—that although complete healing, in spite of the mastoid operation, did not take place, and although it might possibly be due to labyrinthine disease which he had not recognised, yet even if this were the case no serious symptoms had afterwards occurred, and as the result of proper treatment it was very rare that complete cure was not eventually obtained.

Mr. SCOTT, in reply, said there was only one case in their series of labyrinthine disease complicating acute otitis media, though possibly Case 25 may have commenced as such. Some observers said that 18 per cent. of cases of labyrinthine disease were due to acute, and the remainder to chronic conditions. They had not in this paper any figures which would confirm this. The case quoted by Dr. Smurthwaite was not mentioned because it was not operated upon. It seemed similar to their Case 27, for which no operation was performed on the labyrinth. For five days after the operation on the middle ear the patient vomited daily; the headache then followed and the patient died suddenly, without meningeal symptoms, on the ninth day. This seemed to be an important case apropos of Mr. Tod's remarks. He would like to emphasize the continued vomiting as evincing evidence of labyrinthitis which was, unfortunately, not understood, and also the sudden fatal result without antecedent symptoms of meningitis. The nature of this case might have been overlooked; they had had the advantage of performing the post-mortem investigation personally in the following manner: The whole calvarium was removed, exposing the brain and dura mater, by a complete horizontal saw cut. The brain was very tense and there was no fluid on its surface. On cutting through the whole brain horizontally, in situ, a large quantity of cerebrospinal fluid gushed out at high pressure from the ventricles, which remained dilated. The brain was removed in successive slices. In the region of the seventh and eighth nerves there was a distended sac of arachnoid tissue, the fluid there being slightly turbid, but no organisms grew from it. On opening the superior semicircular canal the membranous canal appeared as a bright red filament. Although there was definite pus in the vestibule there was no meningitis in the ordinarily accepted sense of the term. He was convinced that the infection, whatever its nature, extended along the arachnoid sheath and the labyrinth to the choroid plexus of the fourth ventricle. The distension of fluid in the theca of the spinal cord might cause head retraction, because, on lumbar puncture, that retraction ceased. In several of the cases with meningitic symptoms there were no organisms found in the cerebrospinal fluid withdrawn by lumbar puncture. In answer to Dr. Kelson's questions, they felt diffident in stating anything definite at present, owing to apparent contradictory phenomena, but the questions would be followed up. They had not had any success in varying the direction of elicited nystagmus by means of heat. More usually vertiginous patients complained of objects appearing to move towards the sound side. There were two cases in which objects seemed to move to the same side as the lesion. This matter was also being followed up, and they hoped to make a further contribution on the subject.

Deafness resulting from Epidemic Cerebrospinal Meningitis.

By H. H. B. Cunningham, F.R.C.S.I.

One every now and again meets a case of total deafness in which the history obtained is that the patient has recently recovered from an attack of cerebrospinal fever, during which the deafness developed. In view of the fact that the prognosis as regards the hearing in these cases is very bad, and that the methods of treatment at present in vogue do not yield much result, I venture to bring before this meeting notes on two cases in the hope that some light may be thrown on the treatment of this distressing affection.

Case I.

Samuel M., aged 9, seen May 16, 1907, for deafness.

Previous History.—Admitted to fever hospital, March 19, 1907, on the sixth day of the disease, the deafness having commenced on the previous day; the onset took place with headache and vomiting; no delirium nor ear symptoms (other than deafness). On admission there was rigidity of the neck; Kernig's sign was present and a purpuric rash; the deafness remained while in hospital. The blood, tested by Dr. Houston and Dr. Rankin, gave agglutination and a positive opsonic index to the meningococcus.

Present Condition.—When seen the boy was quite deaf, and could not hear when spoken to even in a very loud voice. The deafness was so complete that his father had to write on paper every communication he wished to make to him, therefore tests with the tuning-fork were dispensed with; the watch was tried in contact with the mastoid bone, but the information obtained was quite unreliable. Both tympanic membranes were normal in appearance, neither showing atrophy, nor thickening, nor perforations; they were perhaps slightly retracted. The tonsils were not enlarged and there were no adenoids. The boy was bright and intelligent, and readily did anything he was told to as soon as he understood what was required. No giddiness had been complained of, but when the patient walked across the room he was seen to stagger a little to the left, and he definitely edged away towards the left when walking. A week later there was said to be some slight improvement in hearing. He now complained of giddiness when staggering towards the left. This symptom had probably been present before, but was only noticed since it had been sought for. A week later he was said to occasionally hear the noise made by striking a table, but this could not be elicited at the hospital.

June 6: The giddiness was said to vary in intensity, being more marked in the mornings, when getting out of bed, and when he was excited. He also complained of occasional tinnitus in his left ear.

A week later he heard the hooter at the works one night, and he was said to have heard his mother speak to his father once or twice, and to have understood what was said, but he could not hear the voice at the hospital. The giddiness on waking in the mornings was now lessened, also he could walk in a much straighter line and did not stagger towards the left nearly so much.

June 27: Can walk well now—almost straight, and does not edge away towards the left. Sometimes he is quick at hearing a sound, though unable to interpret it; at other times he does not hear any sound at all.

A week later the walking was about the same, and he could apparently hear carts moving on the road occasionally.

July 25: His general health was very good, and he was bright, and looked intelligent. He did not complain of any vertigo during the day, but still staggered a little on coming downstairs in the mornings. The hearing does not seem to have altered, and he sometimes says he can hear a hooter sounding when none is being sounded.

December 16: Can walk perfectly straight and does not complain of any vertigo now. He is said to occasionally hear trams, and on a few occasions has complained of pain and tinnitus in the right ear, but nothing objective found. He speaks in a natural voice, not in the least elevated, and is quick to do anything that he is told to as soon as he understands what is required, but is quite unable to hear anything that is said to him.

For the first fortnight he was put on strychnine (♏iij.), afterwards on iodide of potash, 5 gr., three times a day, rapidly increased to 10 gr.

CASE II.

Lizzie C., aged 10, was brought to the hospital on June 6, with the history that she had been discharged from the fever hospital three days previously.

Previous History.—Admitted to fever hospital, March 6, 1907, twenty-eight hours after onset of the disease, which commenced with headache and vomiting; no delirium; deafness since onset. On admission there was rigidity of the neck, and Kernig's sign was present. The blood, tested by Dr. Houston and Dr. Rankin, was positive to the meningococcus.

Present Condition.—She is quite deaf and cannot hear anything, every communication having to be written. She walks straight, but suffers from discontinuous vertigo, the tendency to fall being towards either side; there is tinnitus in both ears; the tympanic membranes are normal in appearance, perhaps slightly indrawn on the left side.

A week later the vertigo was only occasionally complained of, the tendency to fall now being in the forward direction; it is more marked in the mornings on rising from bed, when she is decidedly inclined to fall forwards. She was said to have heard a hooter on the previous evening, and to be able to hear the noise of a loud knocking at the door.

June 24: The vertigo has decreased considerably and patient is said to be able to hear words occasionally, but could not hear the voice when tried at the hospital. She was treated with gradually increasing doses of iodide of potash up to 8 gr. three times a day, but was not seen after June 24.

The points of interest in these cases are:—

(1) The vertigo, present in both cases, in each of which this symptom was more marked in the mornings when arising from bed.

(2) The deafness, which practically showed no improvement.

These two cases do not show anything of unusual interest, only the hopeless result of aural complication in epidemic cerebrospinal meningitis. What is the cause of this deafness? The labyrinth, the auditory nerve, or the brain itself may be implicated. Politzer, in his text-book on Otology, quotes several cases, in which purulent infiltration of the labyrinth was found on post-mortem examination, also cases in which degeneration of the eighth nerve was found. At the Manchester Medical Society last year, Orr showed specimens of cranial nerves from a fatal case of cerebrospinal meningitis, in which the reaction of degeneration was present in the third, fourth, fifth, sixth, seventh and eighth nerves, and pointed out that it was only present in the intramedullary portions of these nerves.

The sudden onset, and especially the disturbance of equilibrium, well marked in Case I., would point towards implication of the labyrinth; but Sir Victor Horsley, in his address to the Otological Society in 1905, showed that disturbance of equilibrium may be caused by new growths in the eighth nerve, by lesions in the medulla, cerebellum, and the area of orientation or posterior two-thirds of the temporal lobe. The lesions giving rise to symptoms in these cases, however, are usually deep-seated, and, therefore, are not due to cerebrospinal meningitis; so implication of the auditory nerve or of the labyrinth must be considered the cause of the deafness. Both my cases were treated with iodide of potash, but, so far as the deafness was concerned, without result. Dr. Gardner Robb, to whom I am indebted for the notes from the fever hospital, also informs me that before using Flexner's and Jobling's serum with such success, out of sixty-eight recoveries five were deaf, and since using it, out of twenty-two recoveries one is deaf; so it has not much effect on this complication.

Therefore I would ask: what treatment can we adopt, or in what way can we ameliorate the condition of these unfortunate patients? Should the treatment of the deafness be commenced during the course of the fever itself or, if not—and which more concerns the aurist—what can be done for these cases which, after recovery from the fever, consult the aurist for the deafness?

DISCUSSION.

The PRESIDENT said he thought Mr. Cunningham would find that no treatment was of any use in such cases. In Scotland, every year that he did hospital work he used to see children whose history was that the child had been playing about and suddenly complained of pain in the head. On being put to bed it lay with the head retracted, sometimes with slight delirium and sometimes with loss of consciousness. Often there was vomiting also. After four days the child seemed to recover, and there was vertigo which lasted for some time.

Voltolini, practising in Breslau, published a work on primary inflammation of the labyrinth, and that was soon followed by one by Gottstein, who showed that the cases were the result of abortive cerebrospinal meningitis. He wondered whether his experience in Scotland had been also that in the south.

Mr. YEARSLEY said it would be interesting to know what was the percentage of cases with deafness in cerebrospinal meningitis. In one series of fifty cases reported by Laszynsky there were five of deafness. In two cases one ear recovered, but in the others the deafness was permanent. According to this observer there was destruction due to infiltration of the auditory nerve.

Dr. PRITCHARD said that twenty years ago such cases were common, and then, until lately, there were scarcely any. He did not know whether that was because there had not been so much cerebrospinal meningitis of that type. With regard to the cause of deafness, he always in his own mind concluded that the cause of the deafness was injury to the very soft auditory nerve due to pressure.

Dr. MILLIGAN asked whether any members had experience of lumbar puncture in such cases. Some short time ago he saw a child in whom the diagnosis of cerebrospinal meningitis had been made, and in view of the possible implication of the ear he did a lumbar puncture. Both ears were attacked, and the child became very deaf, and had remained so since. He asked whether lumbar puncture had been done to try and save the auditory nerve.

Dr. GRAY said that there had been a severe epidemic of cerebrospinal meningitis in Glasgow, and he had never seen a case in which recovery from the deafness occurred. What the President had said about the sporadic cases appearing occasionally had also interested him in regard to Scotland. A mother would come with a child aged 3 or 4, and say the child was well until it was aged 6 months or 1 year, and then it got "brain fever," which was the name usually given, and when recovery occurred the child was stone deaf. He would like to know whether the same was experienced in the south.

Dr. BRONNER said that similar cases were fairly common. He had looked upon them as cases of congenital syphilis, as he had seen several get better with inunctions of mercury.

Mr. CUNNINGHAM, in reply, said there had been an epidemic of cerebrospinal meningitis at Belfast for about eighteen months, and of this particular type. A number of cases recovered, but those which had shown aural symptoms remained permanently deaf. Various treatments had been tried previously, but without benefit either to the disease itself or to its aural complication; and unfortunately, Flexner's and Jobling's serum had little effect apparently on the aural complication, though of decided benefit to the patient otherwise. He had no knowledge of lumbar puncture, as he had not seen it tried. He did not think it could be congenital syphilis, as the blood was tested in each case by Dr. Houston and Dr. Rankin, and it was positive to the meningococcus.

Purulent Otitis media with Deep Ulceration of External Meatus in a Case of Secondary Syphilis.

By Adolph Bronner, M.D.

Mr. X., aged 28, has had a sore throat for four weeks. Two weeks ago he had slight earache for one day and since then both ears have discharged, and during the last six days they have been sore and painful. There is perforation of both drums and purulent discharge. The lower halves of both external meati show deep punched-out ulceration with grey surface. There are mucous patches on both sides of the pharynx, and there is a typical specific skin rash and a well-marked indurated chancre in the foreskin.

Ordinary watch heard at 16 in.; tuning-forks nearly normal.

This case is interesting from several points of view. Purulent otitis media is very rare in cases of secondary syphilis of the pharynx. Deep ulceration of the meati is more common in tertiary than in secondary syphilis. In this case the discharge from the middle ears probably irritated the ulcers. Under antisyphilitic treatment and the local application of iodoform and boric acid, and 5 per cent. chromic acid, the local symptoms soon cleared up without causing much deafness. The internal ears were not affected.

DISCUSSION.

Dr. Paterson said he saw a case quite similar, in which the condition of the outer ear was distinctive. Those who saw it readily recognised its nature, as the ulcers were exactly like mucous patches of the pharynx. After treatment, the condition subsided.

Dr. Grant said that though generally unmistakable, sometimes such patches in the ear were very indefinite. In one case it was so indistinct that he overlooked the fact that it was a mucous patch, and the diagnosis was made owing to the discovery of a syphilitic patch on the skin in another part of the body.

Crossed Abducens Paralysis in a Case of Cerebellar Abscess.

By D. R. Paterson, M.D.

E. R., a collier, aged 18, admitted on April 20, 1907, complaining of pain in the right ear and back of head for five weeks; discharge from right ear for two weeks, and still later of vomiting and diplopia.

He was somewhat drowsy, and lay with his head well retracted. He could answer questions intelligently, and stated he noticed the double vision when on his way to hospital. The pulse was 54. There was fœtid discharge from the right ear, which contained granulations. The left ear was normal. Nystagmus, increased on looking to either side was present. There were optic neuritis and distinct weakness of the left external rectus. At the postaural operation, done twelve hours later, the outer antral wall was very thick and hard, and the antrum contained cholesteatomatous debris. Its posterior wall was partly deficient, exposing the lateral sinus and part of the posterior fossa, and it was further cut away as far as the posterior semicircular canal. Through the space so enlarged the cerebellum was explored, and about 1½ dr. of pus evacuated. The sinus was exposed for some distance and appeared healthy, and the middle fossa of the skull was explored with a negative result. A drainage-tube was inserted in the abscess cavity. On April 25 he was much brighter; he could move his head better and slightly flex his neck. He still had diplopia on looking to the left, and a definite squint. On May 5, Mr. Russell Thomas again examined the eyes, and reported there was still double vision on looking to the extreme left and limitation of the movement of the left eye outwards. The optic discs were very swollen and small hæmorrhages were seen. The patient made an uninterrupted recovery, and on his discharge on June 5 the movement of the left rectus was quite normal, though some degree of optic neuritis remained for some time.

DISCUSSION.

Dr. GRANT said he did not see how the abducens paralysis on the opposite side could occur from the cerebellar abscess directly, by pressure. Such paralysis, however, arose from such a number of causes that it was agreed by neurologists to have but little diagnostic importance. The sixth nerve had such a long course, was so exposed, and its associations with the labyrinth were sc numerous that it was difficult to legislate for it.

Mr. TOD said that he had seen a similar case of crossed abducens paralysis occurring in the course of chronic otorrhœa. Its onset was accompanied by slight pyrexia and pain radiating up the affected side of the head. Examination showed a perforation of the tympanic membrane of long standing. There were no objective signs of inflammation of the mastoid process. A diagnosis of intracranial abscess was suggested, but as there were no definite symptoms Mr. Tod advised waiting. Shortly afterwards all the symptoms, including the ocular paralysis, disappeared. Since then (five years ago) the patient has occasionally had slight otorrhœa, but otherwise is quite well. Mr. Tod said he could not express an opinion as to the cause of the paralysis.

Dr. PATERSON, in reply, said he had brought forward the case more or less for the purpose of record. Mr. Tod had shown a case in which both abducens nerves were affected. The present seemed the only case where there was crossed paralysis.

Branchial Sinus leading into External Auditory Meatus; Sinus Excised.

By C. H. FAGGE, F.R.C.S.

FRED M., aged 3½, was sent to me on account of left otorrhœa, lasting two years, which was often foul in spite of persistent antiseptic treatment. His father volunteered the statement that the otorrhœa alternated with discharge from a small sinus situated on the left side of the neck at the level of the hyoid and just in front of the left sterno-mastoid. On examination there was no pus in the left meatus, and the membrane, though partially obscured by a small quantity of wax, seemed normal; the meatus could not be mopped out, as the child was very difficult to deal with. I advised syringing the left ear with carbolic lotion, 1 in 60, twice a day, and excision of the sinus, which I regarded as due to a suppurating tuberculous cervical gland, for it was said that the sinus resulted from the bursting of a lump in the neck before the otorrhœa began. On November 22, 1906, under an anæsthetic, I again examined the ear, but even after mopping the ear out could find no perforation. I then proceeded to excise the sinus, which a probe showed passed upwards deeply into the parotid region. When the stalk was isolated it could be traced up deep to the gland and seemed to be attached to the base of the skull just external to the vaginal plate of the petrous. I now recalled the curious remark of the patient's father, but repeated attempts to pass a fine probe upwards into the external auditory meatus or downwards from this into the sinus failed. The incision was therefore carried up behind the auricle, and the facial nerve was exposed and isolated; the sinus was then defined above the facial nerve, which looped round it, and as the sinus wall was further dissected up it eventually came free, in doing which the external auditory meatus was opened and the point of the probe within the sinus was exposed in the meatus. A small gauze drain was inserted in the lower angle of the wound in the neck, which, except for this, was sutured throughout; the drain was removed in forty-eight hours; primary union resulted. A month later the wound was healed and the meatus and membrane were quite normal.

Dr. Leathem, Pathologist to the Evelina Hospital, reported that the sinus was lined with squamous epithelium, outside which was fibrous connective tissue. The patient's doctor has recently (February 28, 1908) written to tell me that the ear has remained dry and that the sinus has not reopened.

This is in my opinion a case of persistence of the dorsal portion of the second external branchial cleft depression, which in several mammals persists at its lower end in the form of an accessory auricle as first described by Heusinger. Keith notes that in the goat part of the second cleft is marked by an opening and by an auricular appendage, and Bland-Sutton gives a figure of the same condition, but does not record a cervical fistula similar to mine as having been observed in man.

Mr. YEARSLEY said that recently he had a somewhat similar case, on the right side, in a girl, aged about 20; she also had chronic otorrhœa, with a discharging sinus just below the auricle. He explored the sinus and dissected out a small dermoid cyst full of hair, which was attached to the floor of the external meatus.

Epithelioma of Middle Ear (Traumatic?).

By W. MILLIGAN, M.D.

S. W., policeman, aged 45, admitted to hospital complaining of great pain in right ear and continuous discharge of fœtid, blood-stained pus. Ear disease attributed to injury received nine months previously; no history of any ear trouble prior to that date. Large mass of fungating granulation tissue in meatus. Sequestrum in mastoid area felt with probe. Morphia administered. No relief. Microscopic examination of granulation tissue shows a squamous epithelioma. On account of severe pain mastoid opened, fungating mass of granulations scraped out and sequestrum removed; no relief. Ligature of external carotid artery (Mr. H. Lund).

Death two days later. Post-mortem showed middle ear and adjacent mastoid area entirely destroyed. Intracranial abscess (size of a pigeon's egg) found in anterior portion of right temporo-sphenoidal lobe. No erosion of roof of antrum or tympanum.

DISCUSSION.

Dr. PATERSON said that in view of such cases coming into the courts it would be interesting to hear Dr. Milligan's view of the association of injury with the growth.

Dr. MILLIGAN replied that the patient was a policeman, and the injury was caused during the arrest of a man. He had advised the man's wife not to bring the case into court as the causation of the disease was so indefinite he could not give definite assistance. If he had to go into the witness-box he would say that such an injury might cause such a lesion. He carefully cross-examined the patient, who stoutly denied having ever had any ear trouble before the injury. There was free hæmorrhage from the ear, so the membrane had probably been ruptured: the ear began to suppurate and then malignant degeneration appeared to have set in.

Case of Suppuration in the Right Labyrinth; Operation; Recovery.

By MACLEOD YEARSLEY, F.R.C.S.

J. B., AGED 8, was first seen by me at the Somerford Street London County Council Deaf Centre on January 28, 1908. He had been deaf since measles at the age of 1, having only slight vowel hearing. There was very offensive discharge from both ears, often bloody. No static symptoms could be obtained, but no examination was made with a turntable.

He was taken into the Royal Ear Hospital on February 5, and on February 6 I performed the radical mastoid operation on the right ear. The tympanum and antrum were full of very fœtid debris, and the two larger ossicles had completely disappeared. On clearing out the whole cavity a small erosion leading into the external semicircular canal was found. That part of the buttress of bone overhanging the middle ear was carefully chipped away in order to obtain a view of the fenestra ovalis, and that opening was found to be carious at the lower part, the stapes being absent. The following method of dealing with the condition was adopted: the vestibule was thoroughly laid open by removal of the wall below the fenestra ovalis, and its cavity filled with peroxide of hydrogen, which bubbled through the hole in the external semicircular canal. It was then swabbed out with pure formalin, the wound closed and the cavity packed with iodoform gauze in the ordinary way. Beyond twitching of the facial muscles on the right side, especially about the angle of the mouth, which lasted for about forty-eight hours, the boy has not had a bad symptom. The radical operation was performed on the left side on February 13.

Specimens and Photographs of Pathological Conditions found in the Labyrinth.

By Albert Gray, M.D.

(1) Portions of the membranous labyrinth from a case of suppurative disease of the middle and internal ear.

(2) Deposit of calcareous salts in the region of the organ of Corti in a case of otosclerosis.

(3) Deposit of calcareous salts in the vestibule. During life the patient had suffered from giddiness and a slight degree of deafness.

(4) Deposit of calcareous salts in the semicircular canals. During life no symptoms were present.

(5) Deposit of calcareous salts in the canals. No symptoms were present during life.

(6) Membranous labyrinth from a deaf-mute. The results on examination of the labyrinth are negative.

(7) Membranous labyrinth from a deaf-mute. The results on examination of the labyrinth are negative.

DISCUSSION.

Mr. Yearsley asked what was the condition of the middle ear in the deaf-mute specimen.

Mr. R. Lake asked why Dr. Gray called the dark patches calcareous. He thought they were more probably fatty degeneration, and he understood that they had been treated with osmic acid.

Dr. Kelson asked whether the bony labyrinth was correspondingly large. One would not think that such a membranous labyrinth would go into the ordinary bony labyrinth; the membranous labyrinth was usually one-fourth the diameter of the bony.

Dr. Gray, in reply, said it was a normal Eustachian tube, and the tympanic mucous membrane was healthy. The tensor tympani was not atrophied, and its fibres were in a normal condition. In speaking of the size of the membranous labyrinth, he included the endosteum; the endolymph space was very small. In answer to Mr. Lake, he could not say that they were certainly calcareous deposits, but only probably so; they might possibly be urates. He did not care to test the point, as it would spoil specimens which had taken some months of preparation.

Histological Preparations of the Human Labyrinth.

By Sydney Scott, M.S.

(1) Sections of the vestibule with the utricle, ampullary nerves, crista ampullaris.

(2) Sections of the vestibule, saccule, membrana secundaria.

(3) Pathological sections of human labyrinth: (*a*) sections through cochlea in a case of acute diffuse labyrinthitis; (*b*) sections of cochlea in chronic granulating labyrinthitis; (*c*) section of membranous ampulla of external semicircular canal in recent circumscribed labyrinthitis.

Mr. Cunningham paid a tribute of admiration to Mr. Scott for his specimens. He had experienced some difficulty in making similar preparations, but Mr. Scott had promised to make a further communication on the subject at a future meeting.

Specimens of the Temporal Bone illustrating Operations on the Labyrinth.

By C. E. West, F.R.C.S.

(1) Complete ablation of the labyrinth ("extirpation").

(2) Double vestibulotomy, with removal of cochlea.

(3) Double vestibulotomy, without removal of the cochlea.

(4) Inferior vestibulotomy, with partial removal of the cochlea.

(5) Superior vestibulotomy.

(6) Superior vestibulotomy, with complete removal of semicircular canals.

Otological Section.

May 2, 1908.

Dr. PETER MCBRIDE, President of the Section, in the Chair.

The Clinical Pathology of Aural Discharges.

By WYATT WINGRAVE, M.D.

DIVISION OF SUBJECT.

GROSS CHARACTERS OF DISCHARGES :—
    Fœtor, colour, density.

TECHNICAL :—
    Collection, fixing and staining.

COMPOSITION :—
    Cells.—Epithelium ; Leucocytes and Lymphocytes ; Epithelioid ; Myelocytes, &c.
    Bacteria.
    Matrix.

SUMMARY OF DIFFERENT TYPES.

Can any information of real clinical value be obtained by examining an ear discharge? is a very familiar question.

In 1883 Eschle [5] demonstrated the presence of tubercle bacilli in aural discharge. The correctness of his observation has since been proved by many observers. In 1893 Macewen [13] emphasized the importance of a microscopic examination of aural discharges. Five years ago I was privileged to draw your attention to the cytological aspect of aural discharges with special reference to the presence of acid-fast bacteria [34]. I have uninterruptedly continued the research, and now venture to briefly summarize the conclusions in the hope that, supplemented by your experience and criticism, they may help to supply an affirmative answer to the question.

I propose to deal first with the different histological and bacterial elements which are usually found in aural discharges, together with the technical details necessary for their recognition: afterwards to consider the specific characters of the discharge in each variety of disease, with their diagnostic significance. To be of real diagnostic value the specimen must always be obtained from the nearest available point to its source, since it is practically useless to take an antro-tympanic discharge from the meatal aperture, where it is exposed to a wide range of contamination including the epithelial products of that region.

In collecting material, whether for films or cultures, the following precautions should therefore be taken:—

(1) It should be obtained from a point nearest to its probable source.

(2) All contaminations should be avoided, and every instrument should be sterilized.

(3) Its fœtor, density and colour should be carefully observed.

With regard to *fœtor*, the butyric type, which is perhaps most frequently found, is commonly associated with epidermal decomposition such as ungual accumulation and smegma, or with over-ripe cheese. It is doubtless due to changes occurring in epidermal cells in the presence of *Bacillus butyricus*. In aural discharge it is unmistakable and characteristic of a desquamative process, either meatal or antro-tympanic. That it is closely associated with putrefactive bacteria is supported by the fact that sterile cholesteatomata are free from fœtor. Sometimes it has the character of sulphuretted hydrogen, probably derived from the sulphur of the keratin scales. When osseous necrosis or caries exist, a phosphuretted smell is observed, especially in the presence of *Bacillus proteus vulgaris*. Fœtor is rare in the acute forms of aural discharge, but in the chronic it is intimately associated with spirochætes, *Bacillus butyricus*, *Bacillus proteus vulgaris*, streptothrix and various anaerobic bacteria [22].

Density is a feature which may afford valuable information. If serous or "watery" and continuous in flow it is strongly suggestive of cerebrospinal fluid, confirmed by its affording positive evidence of reducing power with deficiency or absence of proteids. If scanty and mixed with flakes it is probably tuberculous or eczematous. In acute exudative processes it may be viscid, glairy or coagulated, according to the nature and intensity of the infection. In chronic cases it may vary from a thin "milky" to a "creamy" or even caseous consistence. It may also be dry and "scaly."

Its *colour* may be of great variety. In all forms the discharge may be mixed with fresh blood. Should this occur in chronic cases it is strongly suggestive of active granulations and polypi; but when the hæmorrhage is retained it may be "rusty" and resemble coffee or "anchovy sauce." The latter colour occurs in connection with malignant disease, while the rusty or coffee colour may be due to cerumen. Many chromogenic bacteria may be responsible for remarkable tints in chronic cases, such as the *Bacillus pyocyaneus* and *Micrococcus cæruleus*. Black or grey granules may be due to aspergillosis, but are far more frequently caused by lead, bismuth and other salts which have been used for instillation or insufflation.

Collection of Material.—The patient should be placed in the usual position for examination alongside a good light. Guided by a speculum, the meatus being carefully cleared of accumulation, a bead of pus is removed from the region nearest to the seat of the disease by a small curette or platinum loop, taking care to avoid contact with any other surface and not to cause bleeding. The drop should be at once transferred to a clean cover-glass, over which another one is placed, then lightly pressed together and separated by sliding. (If separated by *lifting*, the film will be rough or "tacky" and useless for staining.) The film should at once be dried and fixed, either by hot air (not in contact with flame) or by plunging into acid alcohol or perchloride of mercury solution, afterwards washed and dried. Should the discharge be plentiful, it may be collected by a suction pipette of small calibre and then transferred to cover-glass; but if examination is deferred, or if to be sent by post, its end should be sealed in the gas flame. Fixing by perchloride is really only necessary when eosin or vesuvin is used in staining. Cover-glasses are preferable to slides, being far more convenient for manipulation. At least four films should be prepared, while an extra one may be examined "wet" for motile bacteria. A culture may be taken with similar precautions, substituting a fine platinum wire for the curette. It need scarcely be accentuated that everything used should be scrupulously sterilized, and the film must on no account come in contact with a flame for fear of scorching. When antiseptics have been employed and a culture is required, it is better to postpone taking the specimen pending twenty-four hours suspension of treatment.

Staining.—If the staining is to be done by the aid of heat it is better to use a short wide test tube; but if cold, shallow glass dishes are more convenient than watch glasses. The simplest and most comprehensive staining processes are the following:—

(1) Gram's stain—

- *(a)* One per cent. gentian aniline violet in aniline water, three minutes. Wash in water.
- *(b)* Solution of potassium iodide and iodine, half a minute or until of a dark violet colour.
- *(c)* Wash in alcohol until no violet comes away.
- *(d)* Counter-stain half film in weak carbol fuchsin.

  Wash, dry and mount in xylol balsam.

This is an important test, since it at once decides to which group the bacteria belong, Gram + or Gram −.

(2) For tubercle, acid-fast bacilli, and squames. Carbol fuchsin and methylene blue (Löffler), using 24 per cent. sulphuric acid for decolorizing, followed by alcohol. Wash, dry and mount in xylol balsam.

(3) General stain for all bacteria and cells—

- *(a)* One per cent. solution of gentian aniline violet in aniline water for three minutes and wash.
- *(b)* One per cent. methylene blue ("medicinally pure" or "monochromatic") in 0·5 per cent. solution of borax ("borax blue") for three minutes, wash in water; dry and mount in xylol balsam.

These three stains are generally quite sufficient to obtain all information, yet if (3) should prove too deep for the cells use basic fuchsin (1 per cent.) or carbol fuchsin in place of the gentian aniline violet. This stains the cytoplasm a beautiful scarlet, in which the bacteria and nuclei will be well seen when counter-stained with borax blue. It also demonstrates sporulation. Either method affords a most useful combination if carefully carried out, and no one who has been accustomed to work only with nuclear stains can fail to appreciate the beauties revealed by employing a protoplasmic contrast stain. It is most searching for bacteria when used its full strength and time, but for cytology it is better when diluted $\frac{3}{1}$ and with a shortened contact. Solutions must be fresh and filtered. Distilled water only should be employed.

Examination.—This should first be done with a 1 in. or $\frac{1}{2}$ in. lens, which will give a general idea of the larger cells, to be followed by a $\frac{1}{12}$ immersion lens for cytological details and bacteria.

COMPOSITION.

A discharge usually consists of three elements: (1) cells; (2) bacteria; (3) matrix or suspending fluid.

Cytology.—The cells may be divided into two groups: *(A)* epithelial, and *(B)* mesoblastic.

(*A*) *Epithelium.*—Epithelial cells are meatal, tympanic and glandular. The commonest type is the squame, which in the healthy state is strictly confined to the meatus, but in chronic disease invades the antro-tympanic cavity and becomes one of the most striking features of discharge from that region. They are of two distinct kinds, old and young. The old ones stain deeply with fuchsin and resist decolorizing with acid. They are, in fact, acid-fast like tubercle bacilli, their nuclei are wanting or only indicated by a pink area (ghosts). Young or recently formed ones are readily decolorized with acid, and their large oval and round nuclei stain deeply with blue. There are also intermediate varieties whose cytoplasm contains keratin granules which stain violet by Gram and whose nuclei take a faint blue.

This acid-fast property of old non-nucleated squames is one of considerable interest, since it affords not only presumptive evidence of a cholesteatomatous process involving the antro-tympanic cavity—assuming that the specimen was taken from an adjacent spot and not from the meatus—but fragments might easily be mistaken for tubercle bacilli by a careless observer.[1] When taken from the meatus in large numbers they indicate the existence of chronic desquamation, with or without leucocytes. The blue staining nucleated squames indicate recent or existing desquamation.

Tympanic Epithelium.—Normal tympanic epithelium is *only* seen in acute cases. It appears as solitary or grouped spheroidal, cubical and pyriform cells, having a pale clear cytoplasm with a well-defined blue oval or round nucleus, often eccentrically situated. Such epithelium does not appear in chronic discharges, the tympanic lining having become transformed into the squamous or epidermal type.

Gland Epithelium.—Gland epithelium likewise is rarely seen, except in acute cases, and is mostly of meatal origin, since glands are scarce in the middle ear. The cells are variable in size and shape, but can be recognised by a single oval nucleus and slightly oxyphile or yellowish cytoplasm, sometimes staining deeply.

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[1] It is very important to bear in mind this acid-fast property of epithelial squames, since it may lead an inexperienced observer when searching for tubercle bacilli to think that the film is insufficiently decolorized and induce him to prolong the acid and alcohol bath until the fuchsin is either entirely removed from the tubercle bacilli or rendered so faint as to be neutralized by the methylene blue in counter-staining.

The recognition of neoplastic characters is very difficult, but whenever cells are found in closely packed groups and their nuclei are heteromitotic they should always excite suspicion, especially when associated with myelin spheres and red blood-corpuscles.

It will thus be seen that cells may be living or dead, a distinction equally applicable to the mesoblastic group and equally important, since it enables one to determine whether the cells are of recent or relatively remote origin.

(*B*) *Mesoblastic Cells.*—Mesoblastic cells may be conveniently, yet perhaps somewhat arbitrarily, divided into two groups—wandering and fixed.

The *wandering* cells are leucocytes and lymphocytes. *Fixed* cells are mesothelial (endothelial and perithelial), giant-cells and myelocytes. There are also many others, such as plasma-cells, fibroblasts, angioblasts, &c., but their histological distinctions are so undecided that for the present it will be expedient to confine our attention to those whose identity and source can be readily established.

Leucocytes and *lymphocytes* are generally referred to by the comprehensive term "pus cells," but clinical cytology teaches that some discrimination is necessary, since they are unlike one another in structure, function and significance. Whatever views may be held regarding their biological relations, their recognition and differentiation in a discharge are of no slight importance. A fresh *leucocyte* is somewhat larger than a lymphocyte, it possesses a relatively greater area of cytoplasm, which is always granular, the granules being termed oxyphile, basophile or neutrophile, according to their selection of acid, basic or neutral stains, a distinction of importance as blood-corpuscles, but of less value when they occur in a discharge. The nucleus occupies a relatively small part of the whole cell and may be multiple or single; the "polymorphs" are the more prominent in acute discharges, while the mononuclear are present in chronic or the later stages of acute diseases.

The leucocyte of a recent or acute exudate is very sharply defined and the nucleus stains deeply; but it soon degenerates, the outline of the cytoplasm is lost and the nucleus either stains faintly, becomes distorted, or undergoes fragmentation into "myelin" spheres (pyknosis), which may either remain in the cloudy cell, become absorbed by another cell, or escape into the surrounding matrix: a series of changes which indicate its death. One can thus easily distinguish between the living or active and the dead or degenerated stage. As the discharge becomes chronic large mononuclear leucocytes are much more numerous, a characteristic feature of granulating wounds which distinguishes it from the earlier and more exudative process. Their cytoplasm is very granular and stains deeply with gentian violet or fuchsin, while their nuclei are paler than those of polymorphs and more irregular than those of endothelial cells, which possess a very clear cytoplasm.[1]

Each variety of leucocyte may be a phagocyte, a property which can be best demonstrated by means of the fuchsin and borax blue stain. Weak solutions and short exposure are essential, since, if over-stained, the cytoplasmic granules obscure the engulfed bacteria.

The *lymphyocyte* is smaller than the leucocyte; its cytoplasm is scanty, often invisible, or appearing simply as a narrow zone, which is clear when living but becomes faintly granular with degeneration. The nucleus is relatively large, single, round, and stains very deeply with borax blue or gentian violet. In acute exudative changes about one lymphocyte can be counted to twenty or thirty leucocytes, but when the discharge comes from a "granulation" source the lymphocytes are strikingly increased, sometimes being equal in number to the leucocytes.[2] Thus the presence and proportion of these cells afford reliable evidence of the existence of granulation tissue and the nature of the pus-producing process. They possess little, if any, power of phagocytosis.

Mesothelial or Epithelioid Cells.—Of the *fixed* cells epithelioid elements are those most frequently found. The name mesothelium includes endothelial and perithelial elements, since in a discharge it is impossible and immaterial to distinguish one from the other. They are derived from the lining of blood and lymph channels, also from the perivascular spaces of arterioles. They play a prominent and important part in all granulomatous formations, especially that of tubercle, lupus and lymphadenoma, since the greater part of a typical tubercle is composed of them. They are recognized by their large size (nearly twice that of a leucocyte), irregularity in shape, large pale oval nucleus and an extensive clear cytoplasm, which on degeneration becomes granular or cloudy. Although sometimes seen in acute inflammation, their presence in large numbers is strongly suggestive of tuberculosis. They are usually credited with being "cell eaters," but may sometimes contain bacteria; they are, however, less phagocytic than leucocytes, although nuclear fragments of other cells are often seen inside them. It is probably from these cells that "giant-cells" are formed, either by nuclear division or fusion.

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[1] All cells in a film appear larger than when seen in section, because being soft spheres they are more or less flattened in the act of preparation by pressure, however slight.

[2] It must not be forgotten that lymphocytes are much more numerous in infants' than in adults' blood. They are consequently, therefore, often relatively more numerous in the acute suppuration of children. Whether lymphocytes of a discharge are derived from the blood or from the lymph is a debatable point. According to Maximow (9) they may be derived from the lymphoid cells of bone marrow and, growing larger, may become phagocytes. Lymphocytes are also said to become plasma cells in chronic inflammatory processes. Like leucocytes, their nuclei, when old, become paler in staining power.

Myelocytes.—Several kinds of myelocytes are seen in aural discharges, but they may be conveniently divided into two, viz., small mononucleated and large multinucleated. The first variety is difficult to distinguish from a large lympho- or leucocyte; it is, however, somewhat larger, its cytoplasm is clear and more extensive, while the nucleus is single, round, eccentrically situated and larger than that of a lymphocyte. The multinucleated has two or more closely packed nuclei and the cell is two or three times larger than the mononucleated. They are both derived from the red marrow and may sometimes be seen in both acute and chronic inflammation of diploic bone. In cases of primary acute osteomyelitis they are sometimes very numerous, in chronic forms much fewer; the small variety being a striking feature in acute osteomyelitis, while the large multinucleated form occurs chiefly in slower osteoporotic processes, when it is referred to as an osteoclast. In the course of acute infection the thin cytoplasm may contain neutrophile granules.

Erythoblasts.—A much smaller form (erythroblast), whose cytoplasm stains brilliantly with eosin, is not uncommonly found in acute osteomyelitis of infants.

"*Giant-cells*" differ from multinucleated myelocytes by the arrangement of their nuclei, which are generally oval, more numerous and grouped symmetrically round the periphery of the cell, while the nuclei of myelocytes are generally round, fewer in number and crowded together in the centre. They are rarely met with in discharge unless the tuberculous process is in a state of acute exacerbation and rapidly breaking down owing to a supplemental infection. In scrapings taken directly from the diseased spot and ground up with normal saline solution they constitute a striking feature in the film when stained with thionin and eosin.

Bacteria.

There are, perhaps, few organs which present a greater variety of bacteria in their discharge than does the ear, particularly in the chronic forms of disease. In acute stages, while the total number of micro-organisms present may be large, the varieties are generally few as in other regions.

Discharges may be examined for bacteria in two ways: by means of specially stained films and by cultivation on agar, serum, gelatine, bouillon and other media.

I shall refer chiefly to the evidence afforded by films or smears, which, if properly prepared, are of considerable diagnostic value in spite of the occasional difficulty in accepting the identity of a micro-organism solely upon its morphological and staining features.

Cultivations alone should not be relied upon, since they may afford but partial evidence of the bacteria present, and films, if stained by the three methods, may often reveal important bacteria which fail to grow on the selected medium.

From a record which represents a portion only of many years experience I am able to give the following list of bacteria that were specially recorded in the course of 500 examinations of chronic discharges. It is by no means an exhaustive list, since it does not include every bacterial form which was present, but is fairly representative of those bacteria whose identity was provisionally established, and whose frequent occurrence, together with certain clinical and pathological associations, imparts to them a varying measure of interest. Except for the important addition of the spiral forms and their associates the list is similar to the one which I presented in 1903. They are as far as possible arranged in order of relative frequency, the sign + or − indicating a positive or negative reaction to Gram's stain.

Staphylococcus (*albus* and *aureus*)	+
Diplococcus catarrhalis	−
Bacillus proteus vulgaris	−
,, *subtilis*	+
,, *pyogenes fœtidus*	−
,, *butyricus*	+
Torulæ, Yeasts, &c.	+
Leptothrix	+
Bacillus fusiformis	−
Spirochæta refringens (*Spirochæta fœtida*)	−
Streptococcus	+
Bacillus mesentericus vulgaris	−
,, *coli communis*	−
,, *pyocyaneus*	−
,, *tuberculosis*	+
Micrococcus tetragonus	+
Bacillus of influenza (Pfeiffer)	−
Streptothrix	+
Pneumococcus	+
Penicillium glaucum	−
Aspergillus niger	−

In comparison with the chronic varieties my record of *acute primary* cases is somewhat small, partly owing to the great difficulty in obtaining material during the early stages of the affection, a difficulty which doubtless most of us experience in hospital practice, and partly due to my not having kept a record of observation in acute cases until recently. As already stated the bacteria of acute discharges, although often present in large numbers, present but few varieties as compared with the chronic form. In fifty cases of acute primary discharge the following bacteria are recorded :—

Diplococci occurred			36 times
Diplococcus catarrhalis	−	21	
Pneumococcus	+	9	
Meningococcus	−	4	
Gonococcus	−	3	
Streptococci	+		11 ,,
Staphylococci	+		4 ,,
Bacillus coli communis	−		4 ,,
,, *proteus vulgaris*	−		3 ,,
Acid-fast bacillus tubercle	+		2 ,,
,, ,, pseudo-tubercle	−		1 ,,
Bacillus subtilis	+		2 ,,
Spirochæta refringens	−		1 ,,
Bacillus of influenza	−		1 ,,
Hoffman's bacillus	+		1 ,,
Micrococcus tetragonus	+		1 ,,

An arbitrary division into pathogenic and non-pathogenic varieties is attended with many difficulties in the case of the ear, since the potentiality for mischief in this organ which so many reputed saprophytes possess renders such a classification unwise. As our immediate purpose is the subject of their identification and significance, they will be dealt with in groups which possess some special interest, irrespective of any orthodox classification.

Ear discharges are particularly rich in a variety known as *acid-fast*, a term which is applied to those bacilli which retain their stain (basic fuchsin) after washing in sulphuric acid. By further treatment with alcohol they all give up this stain to a greater or less extent, with the exception of tubercle and leprosy bacilli, which are therefore alcohol- as well as acid-fast.

Although this acid-fast property was at one time believed to be monopolized by tubercle, it is now recognized that a very large number of bacteria also possess it; but since many of them do not equally and constantly respond to the alcohol distinction, they thus possess a technical interest which is in some of them perhaps disproportionate to their clinical importance. Morphologically they fall into three distinct groups: (1) rod forms; (2) streptothricial; and (3) clostridial.

The first, or pseudo-tubercle group, embraces many bacilli which possess some morphological resemblance to tubercle bacilli, but are much more readily decolorized by either a longer exposure to acid or by alcohol, features which are characteristic of the smegma bacillus. They vary from 2·5 μ to 4 μ in length and occur in very numerous "clumps" or groups. Each cluster is, however, composed of far greater numbers than that which an ordinary tubercle group presents. They stain more evenly, are not beaded, and are definitely Gram negative. They occur in company with the *Bacillus butyricus*, acid-fast squames, and are generally attended with considerable fœtor. In this group should be included the *Bacillus subtilis*, which, although not recognized as an acid-fast organism, does sometimes exhibit that property in chronic aural discharges. It is easily recognized, being much thicker and longer than the tubercle bacillus. Further, it has a well-marked equatorial endospore and often occurs in chains. The endospore generally retains the fuchsin after an acid bath, but yields it after prolonged exposure to alcohol.

There are often many short bacillary forms which may show a slight acid-fast property should the acid bath exposure have been too short; but they can be easily differentiated in other parts of the field by their selection of the blue stain.

Streptothricial forms are recognized by their characteristic grouping in the shape of closely packed "felted" masses of branched filaments. But when solitary or in short segments their beaded appearance strongly resembles tubercle bacilli [1]. They are, however, more slender, and, although often Gram positive, are readily decolorized by alcohol. There are also many streptothrices which are not acid-fast, but all are botanically allied to the tubercle bacillus [1] [22].

Clostridia are easily recognized by the presence of a large, deeply staining, equatorial spore. This bacillus is somewhat short, thick, swollen in the centre, and more readily yields its fuchsin to alcohol than any of the others. This is an important group, since it includes the *Bacillus butyricus*, which is one of the micro-organisms responsible for the production of butyric acid, so closely associated with the acid-fast property and with fœtor.

In addition to the foregoing there are many other forms of bacilli which may assume an acid-fast property in the ear, but such a power does not seem to be possessed by any coccal form. Apart from their differential diagnosis from tubercle, acid-fast bacteria are chiefly of interest from their frequency and number, and from what is known of streptothricial infection of other organs the association of this micro-organism with aural discharge is not without significance.

What is the explanation of this acid-fast property?

After many failures to cultivate acid-fast bacilli aerobically I felt that they might possibly be anaerobes, for it was pointed out by Pasteur, and in a recent article by Rist [22], that putrefactive changes in septic otitis were chiefly due to anaerobic bacteria. However, upon exposing films of bacteria (cultivated from ear discharge) to butyric acid, I found that several forms acquired acid-fast qualities in degrees which varied with the duration of exposure to the acid. As San Felice [25] had found that certain bacilli could be rendered acid-fast by cultivation in butter and lard, I added butyric acid to the culture media and succeeded after several attempts in obtaining bacilli which possessed acid-fast property in varying degrees. Butyric acid was selected, from the striking resemblance which its odour bears to that of fœtid aural discharge, a feature which was emphasized by the smell of a bottle of cerumen and meatal accumulations which I had collected for experimental work.

A case was recently reported by Rolleston and Higgs [24] in which a diagnosis of tuberculosis was based to a certain extent upon the presence of acid-fast bacilli in the sputum, but which was proved by the necropsy to be one of typical squamous epithelioma of the stomach. The bacilli upon which the diagnosis was made were subsequently found in groups on the surface of the growth and in the substance of sections. Individually they were unusually long, not beaded, and resisted decolorization by alcohol for two hours. Cultivation attempts were unsuccessful. The case is interesting by reason of the association of acid-fast bacilli with squames undergoing keratinoid changes, and also by their occurrence in a region rich in butyric acid, conditions which are well marked in chronic aural discharges.

In a previous discussion Dr. Jobson Horne [10] suggested cerein as being the substance responsible for the acid-fast property. However that may be in the ear, it cannot hold good in the stomach, skin, lungs, cysts, &c., where cerein does not occur. My experience so far indicates that there are two distinct kinds of acid-fast bacilli—those which are so *naturally*, and those which become so *artificially*, or at all events as a result of their environment. The acid-fast property of the tubercle bacillus and the dead squamous epithelium of cysts and cholesteatomata is essentially intrinsic, while that of the bacteria under consideration is most likely fortuitous or extrinsic. Although endospores are often strongly acid-fast, it is interesting to note that coccal forms, *e.g.*, staphylococci, streptococci, diplococci, &c., never exhibit such a property naturally or artificially, even after prolonged exposure to butyric acid.

In attempting to explain the nature of this property the leading features may thus be summarized:—

(1) Acid-fast bacteria are present in putrefactive conditions which are attended by the presence of butyric acid, *e.g.*, ear, stomach, intestines, skin, &c., and also in butter and cheese.

(2) The acid-fast characters may be produced artificially by butyric acid.

(3) The acid-fast bacteria are generally associated also with the presence of squames and a distinct and characteristic fœtor.

The next group, important both clinically and histologically, consists of four diplococci: (1) *Diplococcus catarrhalis*; (2) pneumococcus (Fraenkel); (3) meningococcus; (4) gonococcus.

Perhaps around none other group of bacteria has there been such a conflict of opinion both clinical and pathological. On the one hand we are told that 60 per cent. of healthy people carry pneumococci [11] in their throats, yet our experience teaches that that micro-organism is not only responsible for serious aural troubles but also for meningitis. It should therefore be considered a conditional parasite [11], and, although harmless in the mouth, of serious import if found in the ear.

It must not be supposed for one moment that it is possible to unequivocally identify a specific diplococcus in a film preparation taken straight from a discharge: such evidence must only be considered as *presumptive*; a *positive* diagnosis requires confirmation by culture in artificial media, under the most precise bacteriological routine. Still, notwithstanding their great mutability in size and shape, evidence of no small value is obtainable by simple and rapid methods.

The film should first be obtained by Gram's method, and if *positive* it is probably a *pneumococcus*, since the other three are Gram negative. Should it be capsulated, oval or lanceolate, like two candle flames placed base to base, it is presumably the pneumococcus of Fraenkel. Too much importance must not be attached to the size nor upon the fact that it may be arranged in short chains of pairs (torula chains), since staphylococci and streptococci may appear as pairs also.

The pneumococcus is not easy to grow, the best medium being blood-agar at 37° C., when it appears as minute, discrete, translucent "dewdrops," very slightly elevated.

The colonies are accompanied by a marked change in colour of the surface medium, shown by a transformation of the oxy- into methæmoglobin, a property described by Eyre [6] as a pathognomonic feature. Further, it produces an acid reaction with dextrose, lactose, levulose, galactose and maltose. It will not grow on gelatine at 20° C., and when artificially grown has no capsule and is often moniliform.

The remaining diplococci are Gram −, but the commonest of all of them is the *Diplococcus catarrhalis*, even more so than the pneumococcus. It is by far the largest and occurs as a well-defined spherical diplococcus except when dividing, it then being "chestnut-shaped." It is not usually encapsuled, but often occurs in chains. It will grow on almost any medium, especially on gelatine at 20° C.; in fact, it is the only Gram − diplococcus which does so [31], [29], [8]. Finally it produces an acid fermentation with all the sugar media and grows well on nasgar at 25° C.

My own experience is that Gram + diplococci are of much less frequent occurrence than Gram −, not only in acute, but also in chronic inflammation and in operation wounds of the ear.

Should the diplococcus not grow on gelatine and be Gram − it is either *meningococcus* or *gonococcus*. Morphologically the *meningococcus* is generally very small, but in cultures may assume a large size. When young it is facetted or "chestnut-shaped"; older forms are spherical. It is not encapsuled. It takes carbol thionin readily, while its occasional intracellular position is not peculiar. The best medium for its culture is nasgar at 37° C., on which it grows as smooth translucent circular discs, and is the only Gram − diplococcus to do so. It causes acid fermentation with all saccharine media except sucrose. Whether this is the micro-organism responsible for epidemic cerebrospinal meningitis there still seems some doubt.

The *gonococcus* is almost persistently facetted, reniform, or chestnut-shaped. Its intracellular character is marked by four or more occurring in every fourth or fifth leucocyte. In size it is smaller than *Micrococcus catarrhalis*, but generally larger than pneumococcus and meningococcus. It grows only on blood-agar and nasgar, the latter at 22° C. Finally it causes acid fermentation with glucose and galactose only.

While fully realizing the difficulties incidental to the identification of any micro-organism, especially in the case of a diplococcus, it is not too much to say that a reasonably reliable and presumptive diagnosis may be obtained from the morphological evidence of a well-stained film. Confirmatory evidence by culture, however, must always be employed in the event of serum or vaccine treatment being entertained. Much assistance may be afforded by attention to the following points:—

(1) Carefully note the Gram reaction.

(2) A chain of moniliform grouping is not necessarily streptococcal.

(3) That encapsulation is not always a reliable distinction, and that any variety may be intracellular.

(4) That too much reliance must not be placed upon the size and shape of individual diplococci, since great variation occurs in the life history of each type.

(5) That it is better to be guided by average than by solitary examples, to remember that every diplococcal form in the ear should be regarded with suspicion, and that the smaller it is the more serious its significance [11].

Spirochætæ, &c.—I will now direct your attention to a group of micro-organisms which, although familiar in the throat and other situations, have not hitherto been identified as attendants of aural lesions; and it is only within the last eighteen months that I have established their intimate association.

Spiral forms similar to if not identical with *Spirochæta refringens* (*Spirochæta buccæ, Spirochæta dentium*) occur in large numbers in chronic and occasionally in acute discharges from the middle ear, invariably accompanied by spindle-shaped bodies known as *Bacillus fusiform* (*Bacillus hastilis*, bacillus of Vincent), &c. They are not readily seen in thick discharges unless deeply stained, being either hidden by the matrix granules or fail to be visible from their weak affinity for ordinary stains. Since writing my first account of them [33] I find that they can be readily demonstrated by the following simple method: After fixing by heat or alcohol the film is placed for ten minutes in a 1 per cent. solution of gentian aniline violet. It is then washed in water and passed through Gram's iodine solution for one minute, washed in water, and finally counter-stained for five minutes in a carbol fuchsin (0·5 per cent.). Borax blue may be used instead of fuchsin as it specially selects the fusiform bodies. Azure blue and Giemsa's solution demonstrate both forms, but they are very expensive and not so reliable as the first method, which is an ordinary Gram without the alcohol bath, this being contra-indicated owing to both bodies having a Gram negative reaction.[1]

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[1] For these and other faintly staining bacteria I have found that potassium permanganate (1 per cent.) makes an excellent substitute for iodine, in combination with gentian aniline violet.

These bodies are nearly always associated with a peculiar fœtor differing somewhat from the butyric smell, being more like that of strong Roquefort cheese, a peculiarity which was specially noticeable in the pus from cerebral abscesses, which contained them in large numbers.

The *spiral* form varies considerably in size and shape. Usually it is long, slender and undulating, with from four to six coarse unequal curves, and pointed at each end. When blunt it is probably due to fracture. Not infrequently, especially in a dense matrix, it may be straight, looped, curved or coiled. It is apparently homogeneous throughout, of a lavender or pale bluish colour when small, but when larger and thicker it stains more deeply. In thin films they may be seen even unstained by reason of their refractile property. They occur singly or in thick felted masses. Compared with the rigid corkscrew curls of the *Spirochæta pallida* this spirochæta more resembles an eel or a whip-lash, having coarser, fewer, and less regular turns. I have never seen it segmented like the *Spirochæta sputigenus* of Miller or the comma bacillus of Koch. It is evidently an undulating form of a straight filament, and its variation in shape depends upon its age, the density of the medium, and the form it possessed at the moment of fixation. The *fusiform* body is also Gram negative, but as a rule takes basic stains more readily than the spirals and is always a more prominent feature in films. It also varies in size and shape, being from 5 μ to 25 μ in length by 1 μ to 3 μ in thickness at its centre, from which it tapers to each extremity. It occurs singly or in pairs "end to end," and is often marked by two or more darkly staining granules with a clear interval near the equator. Occasionally shorter and blunted forms may be seen, but it can scarcely be said to resemble a bacillus. Not infrequently it is bent like a "boomerang."

So far, I have not succeeded in growing either form aerobically; small fusiform bodies were, however, cultivated by deep stab inoculation in agar, but not the spiral form. Both, however, have been grown anaerobically on ascitic agar by Tunnicliffe [32], the fusiform bodies appearing on the second or third day followed by spirals on the fifth.

From the fact that these two bodies are almost inseparable companions, that intermediate forms can be seen in films and their transition apparently traced, it is reasonable to assume that they represent phases in the life history of one organism. They are evidently not bacilli, but belong to the Treponema family, and although occurring as saprophytes in connection with decomposition processes in many parts of the body, in view of the fact that I found them in large numbers not only in the mastoid antrum but also recently in cerebral and cerebellar abscesses, under Mr. Stuart-Low's care, I feel that such an association affords strong presumptive evidence of their possessing a pathogenicity not quite in harmony with their saprophytic reputation.

In many cases, too, of chronic disease of the nasal accessory sinuses I have found them to be the predominant micro-organism. Their occurrence in pyorrhœa alveolaris is well known. During the last eighteen months they have been present in about 30 per cent. of aural discharges of the chronic fœtid type and often afford such a striking feature in the film as to suggest that it must have been taken from the mouth. They have been found in hospital gangrene, acute and chronic forms of tonsillitis, noma, vaccine pustules, cancer, smegma, balanitis, vaginitis, venereal warts, &c. [14], [7], [27]. The *Spirochæta pallida* of Schaudinn I have not yet seen in aural discharge.

Leptothricial forms are frequently associated with the foregoing spirals, in company with torulæ, yeasts, &c., which are generally grouped as "throat" organisms. They are specially numerous in the profuse non-purulent discharge referred to later.

Many varieties of *streptococci* are described, but in aural discharge they may be considered under two types: (1) the *longus* (*Streptococcus pyogenes*), which occurs in long chains; (2) *Streptococcus brevis*, which occurs in short chains. The *Streptococcus longus* is held to be the pathogenic form, while the *Streptococcus brevis*, which is commonly found in the mouth and throat, is said to be without pathogenic properties.[1] They are both Gram +. The individual cocci are somewhat larger than the staphylococcus, they often appear diplococcal when in chains, but their Gram reaction affords a reliable differentiation.

There is a minute variety, the *Streptococcus conglomeratus*, which, though not common in ear discharges, is by no means rare in acute tonsillar affections, and should therefore be looked for in acute suppuration of the middle ear, since it is said to possess considerable virulence [18].

A *Streptococcus mucosus* has recently been described by Schottmüller [26] as responsible for a large proportion of cases of acute suppuration of the middle ear. It is said to occur in short chains like *Streptococcus brevis*, but composed of diplococcal elements enclosed in a delicate capsule which can be demonstrated by acid thionin stain. Like the other streptococci and the pneumococci it is Gram +, but differs from the latter in its shape. Dr. Dundas Grant drew my attention to its literature a short time ago, but my experience in its search, so far, has not enabled me to form any definite conclusion as to its occurrence in this country.

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[1] Marmorek, however, holds that length of chain is variable, and Widal has shown that the non-pathogenic forms from the mouth, when cultivated with *Bacillus coli communis*, become pathogenic [18].

Staphylococci are easily recognized by their grouping. They are Gram + and smaller than streptococci, but whether aureus, albus or cereus cultivation alone can decide. Their pyogenic powers are fully established, and they are the most easily grown of all the pathogenic bacteria.

The *Bacillus proteus vulgaris* is of interest chiefly from its frequent occurrence in chronic discharges, being present in quite 50 per cent. of examinations. Like the colon bacillus it stains with difficulty unless previously treated with iodine or potassium permanganate. It is Gram – and about 3 μ in length (with a central construction), but may grow into long leptothricial threads. It is nearly always associated with fœtor and has the reputation of being a powerful ptomaine producer [11].

The *Bacillus subtilis* is very common and one of the largest bacilli found in the ear, being from 4 μ to 6 μ in length. It is easily recognized by its size and equatorial spore so well differentiated by the fuchsin and borax blue stain. It is Gram + and occasionally exhibits the acid-fast property. It is also associated with fœtor.

Streptothricial forms are not uncommon, and have already been referred to under the acid-fast group; most of them, however, do not possess this property, which is most probably fortuitous. They are all Gram + and also Neisser +, and can be recognized by their peculiar grouping as felted masses of thin beaded and often branching filaments, showing irregular staining. The characteristic clubs of *Actinomyces bovi* are not seen.

The *bacillus of influenza* (Pfeiffer) has the reputation of playing an important part not only in acute exacerbations of middle ear suppuration, but also in primary attacks, an association first demonstrated by Pfeiffer himself. Owing to its feeble staining reaction it is easily overlooked, but if treated with gentian aniline violet and iodine or potassium permanganate it is readily seen in acute cases, especially those complicated by osteomyelitis. It appears in irregular groups or "flocks" of minute straight bacilli about 1·5 μ in length, sometimes in pairs but never in chains. It is Gram – and also strongly selective of fuchsin, a striking feature when used in combination with borax blue.

The subject of bacteria must not be dismissed without reference to torulæ. They are by no means uncommon in chronic cases, but are probably of but slight pathogenic importance. They can be easily distinguished from cocci by their large size. Although spherical when fully developed they often appear like "double chestnut," or in chains. I recently examined a case of acute discharge with a large perforation (about the twenty-first day) in which they were present in large numbers without any other bacteria. The patient, a youth, was also the subject of tonsillitis and naso-pharyngeal trouble, his throat being infested by the same micro-organism, which was shown in both films and cultures.

Chemically and microscopically the *matrix*, or fluid part, of a discharge varies considerably according to its origin. In acute cases at first it is usually clear or slightly opaque, and contains mucin and globulin. Mucin predominates in the catarrhal type and is converted into a homogeneous readily staining film by alcohol, but is precipitated as minute granules if fixed by acetic acid. Globulin, when heated or fixed by alcohol, becomes stringy. In acute membranous or plastic exudates fibrin is formed spontaneously from fibrinogen in the form of regular interlacing filaments, which stain deeply by gentian violet.

In chronic as well as in the later stages of acute cases, the matrix is generally opaque owing to granules of various size and shape, which stain readily and obscure the film. The smallest are derived from degenerated cytoplasm, while the larger and more deeply staining particles are nuclear derivatives. Others may be from broken-down bacteria.

When squamous epithelium is present the minute particles are very numerous, being probably keratin granules, and stain deeply with Gram's reagent. Mixed with them are highly refractile fat spheres selecting osmic acid and sudan iii. When in the leucocyte they constitute "sudanophiles."

Cholesterin crystals, in the shape of flat rhombic plates and feathery crystals of fatty acids, are characteristic of old desquamative changes as in cholesteatomata. Bone particles are easily recognized by their hard grittiness and by the breaking of cover-glasses in preparing the film, but are by no means common. Cerein is recognized by its bright yellow spheres or masses in unstained films, and by its selection of fuchsin and eosin when stained. The more acute the process the clearer the matrix, while the older and more chronic the discharge the more opaque and granular is the film.

In some acute primary and acute exacerbations coarse spirals, similar to Curschmann's in asthma, are occasionally seen. Although smaller than those occurring in sputum, they conform to the classical description in possessing a deeply staining core, surrounded by a pale transparent envelope.

SUMMARY.

Acute suppuration of the middle ear in its mild (catarrhal) form is characterized by sharply defined leucocytes (polymorphs), very few lymphocytes and tympanic epithelium, singly or in clusters. A Gram − diplococcus (*Micrococcus catarrhalis*) most frequently occurs, occasionally associated with mouth organisms such as spirochætæ and torulæ.

In the severe or suppurative type leucocytes and erythrocytes predominate at first with a few lymphocytes. Later the erythrocytes disappear while large mononuclear leucocytes become well marked on about the third or fourth day. Tympanic epithelium occurs early but disappears until healing commences. The cytoplasm of the leucocyte becomes granular and ill defined, while the nucleus stains faintly and is distorted and fragmented towards the second week. In infants lymphocytes are much more numerous than in adults.

Many bacteria are found in acute discharges, including "throat organisms," but the more prominent are *Diplococcus catarrhalis*, *Diplococcus pneumoniæ*, *Streptococcus brevis* and *longus*.

Acute external otitis may occur in several degrees from an acute desquamative process involving the superficial structures only, characterized by nucleated squames, gland epithelium and leucocytes, to abscess or purulent cellulitis involving the deep structures, when leucocytes and lymphocytes will be abundant, accompanied by streptococci, diplococci, staphylococci and also rarely gonococci.

When the disease assumes a chronic form the discharge is "watery," lymphocytes and leucocytes being few or wanting. Epithelial squames are plentiful, and among many varieties of bacteria the *Penicillium glaucum* is prominent.

The conditions responsible for chronic discharge from the middle ear —comprehensively termed chronic suppuration of the middle ear—are so varied that pathological accuracy demands some differentiation.

As most frequently happens "granulating tissue" is responsible for the pus. Evidence of this is afforded by the presence of leucocytes of all kinds, large, small, mono- and polynuclear, normal and degenerated, but especially by *lymphocytes*, which are very numerous, while epithelial cells are not uncommon. Bone disease may be marked by myelocytes or osteoblasts.

Cholesteatoma is indicated by the presence of closely packed acid-fast squames with or without bacteria. This latter may appear to be an unnecessary distinction, but it is really one of great importance, especially when the cells are of antral source, for a septic cholesteatoma in that situation affords a stronger reason for radical measures than a non-septic one; an interpretation which is amply supported by examination of antral contents removed by operation.[1]

Among the many varieties of chronic discharge my experience has taught me that there is one which deserves special attention. It is generally very profuse, intermittent, extremely fœtid, opaque and of the consistence of cream. On examination it is found to be entirely free from cells, either epithelial or leucocytic, but consists entirely of throat organisms in an albuminous matrix. Strictly speaking it is therefore not true pus, but merely a polymicrobic emulsion. It appears as if the imperfectly drained and ventilated antro-tympanic cavity had assumed the rôle of a cultivating chamber or "septic tank" containing bouillon in which different families of bacteria abundantly flourished.

By the term "throat organisms" is meant a group of bacteria which are nearly always to be found in the mouth and faucial area, either in health or disease, but do not occur in the healthy ear. It includes *Spirochæta fœtida*, *Bacillus fusiformis*, leptothrix, *Bacillus subtilis*, *Bacillus proteus vulgaris*, *Penicillium glaucus*, together with a large variety of moulds and yeasts which may be potentially pathogenic, but for the time are leading a saprophytic existence in the throat.

In this group of cases, which is by no means a small one, a highly "septic" state of the mouth, fauces or nasal cavities invariably coexists, the commonest form being pyorrhœa alveolaris and chronic lacunar tonsillitis, affections which are probably not only responsible for the original infection, but also for repeated renewals of the aural trouble.

With such a discharge, whose most striking feature is the large number of spiral and fusiform bodies with practically no leucocytes or lymphocytes, the existence of an active granulation surface may unhesitatingly be excluded. There is a passive yet highly septic cavity which calls not only for active aural measures, but also for attention to the original source of infection. It is the differentiation and identification of such a condition as this that will fully repay the extra trouble of a microscopical examination of the discharge. Such cases further illustrate the necessity for a bacteriological examination of the naso-pharynx, which I have found in healthy subjects to be sterile, but invariably septic in all acute and chronic infections of the antro-tympanic cavity.

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[1] While examining antral contents removed by the mastoid operation several specimens of infection by mycelial threads have been met with, the squames being mixed with a beautiful labyrinth of straight, tortuous and often branching filaments of three distinct types—leptothricial, streptothricial and mycelial.

It is important to note that in *acute exacerbations* of the chronic suppurative form the discharge somewhat resembles the primary acute. Freshly exuded leucocytes prevail with a diplococcus, Pfeiffer's bacillus or streptococci, and diplococci. Lymphocytes are often numerous, and myelocytes may be seen should there be any bone complication. In the course of a few days mononucleated leucocytes become plentiful, with a few more lymphocytes and nucleated squames.

In tuberculous examples of this group there will also be present the specific bacilli. But tuberculous discharge, when *chronic*, is of a very distinct type. It is thinner or watery in character, with white granules or flakes. Lymphocytes are plentiful, with large epithelioid and even "giant-cells." Leucocytes are far less numerous than in non-tuberculous, except during an *acute* period due to supplementary infection, when the discharge is much denser and distinctly purulent in type. The presence of minute amorphous granules and "bone grit" is also a marked feature of tuberculous discharge. Giant-cells are rare unless the specimen be taken directly from its source. As in tuberculous sputum, *Micrococcus tetragonus* is a frequent attendant. When the process is mixed, as usually happens, many varieties of bacteria will be present and attended by marked fœtor.

In *acute osteomyelitis*, the discharge contains, in addition to leucocytes and lymphocytes, considerable numbers of large mono- and multinucleated myelocytes, which are easily distinguished by their size, shape, nuclei and staining. There may be many bacteria present, but few varieties, diplococci and streptococci predominating.

Chronic desquamative external otitis may be either moist or dry. In the former many bacteria are present, with but few leucocytes and many squames, old and new. The commonest bacteria are staphylococci, *Bacillus butyricus*, *Bacillus proteus vulgaris*, mycelia and torulæ. In the dry form only scales occur with *Aspergillus niger*, *Penicillium glaucum*, *Bacillus butyricus* and *Bacillus proteus vulgaris*. In every form acid-fast squames are a very prominent feature.

CONCLUSION.

While fully conscious that these few observations merely touch the fringe of the subject, I trust that they will be sufficient to demonstrate the promises and possibilities which are afforded by an examination of aural discharge. That such evidence is of real and practical value I have now less hesitation in advancing than I felt five years ago, being reinforced by experience gained in the interval, upon an abundant supply of material, rendered doubly valuable by the hearty coöperation and interest of my colleagues.

That clinical pathology is now becoming justly appreciated is shown by the many useful publications which are available. Sputum, blood, urine, vomit, fæces, cerebrospinal fluid, and discharges from many sources are most exhaustively dealt with, but aural discharge—one of the most prominent features of our work—has been practically ignored.

I therefore venture to submit that a careful and systematic examination of an aural discharge will reveal much of the nature of the morbid process which it accompanies; that it should be cytological as well as bacteriological, and that it should constitute an essential part of our clinical routine.

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DISCUSSION.

Dr. Jobson Horne expressed his thanks for the paper, which he considered approached the subject of otology from the right end. He knew by experience the amount of time and labour entailed in work such as the paper dealt with. It would therefore be readily understood that any criticism he might make was not intentionally destructive; on the contrary, he was desirous of criticizing the paper in the most kindly manner possible, and his criticism would be directed to a more practical way of diagnosing cases. The mere staining of films and the examination of them under the microscope taught little more than a clinician could learn from the case itself. It was necessary to resort to cultivation on plates as well as in tubes and animal experimentation. The preparation and staining of films in the application of bacteriology to the elucidation of aural diseases was not sufficient. The manner of collecting the material, as Dr. Wingrave pointed out, was a most important point. The bacteriological technique required in aural cases was at times most difficult, hence the discrepancies in results. When the ear was already the seat of discharge, of course a bead of the fluid could be removed by a small curette or platinum loop, as Dr. Wingrave had mentioned. When the drum was intact very valuable information could be obtained by adopting a procedure which he (Dr. Horne) referred to a few years ago when the British Medical Association discussed the means of preventing acute suppurative disease or acute middle ear disease from becoming chronic. He pointed out how the advisability of incising the drum in a doubtful case could be decided by obtaining a bead of fluid from the middle ear by plunging a fine pipette into the bulging portion of the drum and subsequently submitting the fluid thereby withdrawn to bacterioscopic examination. The removal of the fluid in such a manner could in no way be detrimental to the patient, and even if the examination were negative, the puncture would have relieved tension and pain. If the examination gave positive evidence, then incision of the drum might be proceeded with and the case dealt with on surgical lines, a course of treatment one might be reluctant to adopt in the absence of any definite evidence of the nature of the case. Dr. Horne agreed with Dr. Wingrave that cultivations alone could not be relied upon. He agreed that films, when stained by the different methods, would often reveal important bacteria which might fail to grow on the medium selected. Nevertheless, cultivations would often give valuable evidence not obtainable from films, as, in differentiating acid-fast bacilli, the temperature at which they grew was helpful. It was impossible for any one of them in the short time at their disposal to discuss all the points raised in the paper. One could only consider some of the broad questions at issue. The classification of bacteria into two groups, according to their giving a positive or negative reaction to Gram's stain, Dr. Horne considered did not mark any useful advance in the practical application of bacteriology to otology. Such a classification had already been worked out. The Section approached the subject from the standpoint of practitioners, and he thought the useful classification was into pathogenic and non-pathogenic organisms. He fully agreed with Dr. Wingrave that such a classification was attended with many difficulties in the case of the ear, but those were just the difficulties which they were desirous of surmounting. Questions such as whether the normal tympanic cavity contained bacteria or was devoid of germs, and further, if it did contain bacteria, the conditions under which they remained quiescent or became active, and also when they assumed a pathogenic or a non-pathogenic rôle, still called for investigation. Dr. Wingrave, in touching upon the part played by the diplococci in aural diseases, had stated that the pneumococcus, although harmless in the mouth, was of serious import if found in the ear. A few years ago he (Dr. Horne) conducted experiments to find out how often the organism referred to was met with in the presumably normal ear. For those experiments obviously he could not avail himself of living subjects, and so conducted experiments on subjects in the post-mortem room. With all bacteriological precautions, paracentesis was performed by a fine pipette immediately after death in cases with intact drums, and the fluid withdrawn was bacterioscopically examined, cultured, and animal experimentation resorted to. The results were most interesting. They showed that bacteria did exist in the normal ear, that at times they were pathogenic and at other times non-pathogenic, and experiments further went to show that there was a large field for research in this direction. Dr. Wingrave had been good enough to refer to his (Dr. Horne's[1]) work upon the pathogenesis of tuberculosis in the ear and on the acid-fast property of bacteria met with in that organ, and to give to him the credit of having suggested cerein as being the substance responsible for the acid-fast property. Dr. Horne said that he had pointed out the possibility of a bacillus becoming artificially acid-fast from its environment in the ear. He referred to communications to the British Congress on Tuberculosis by Dr. Moeller[1] and Dr. William Bulloch.[2] Dr. Horne referred also to the list of the acid-fast bacilli and streptothriceæ in the Descriptive Catalogue of the Museum of the same Congress, pp. 1 to 11. Dr. Horne, in conclusion, repeated his gratitude to Dr. Wingrave for bringing the matter forward. The bacteriology of aural diseases was, perhaps, in one sense only too practical, whilst in another sense it was not practical enough. The subject was one that had not received the attention it should have received, especially from the junior members, who would find bacteriological training of great use, not only in clinical diagnosis but also in the surgical treatment of their cases.

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[1] "The Clinical Diagnosis and Surgical Treatment of Tuberculosis of the Temporal Bone, considered with reference to the Pathology and Morbid Anatomy," by Jobson Horne, M.D., *Brit. Med. Journ.*, 1903, ii., p. 77.

Dr. WYATT WINGRAVE did not agree with Dr. Jobson Horne's view that the mere examination of films taught little more than a clinician could learn from a case. The object of his paper was to show that a simple method of information which was employed in connection with every other organ of the body was of like value when applied to aural discharge, and he fully realized the necessity and scientific value of further supplemental research by means of plate cultures and animal experiments, but such research was only available to the special expert. His hesitation in adopting an arbitrary classification of bacteria into pathogenic and non-pathogenic was fully justified by Dr. Horne's experience with the pneumococcus, although such evidence afforded by the cadaver was very unreliable. He could not accept the view that any derivative of ceruminous glands was responsible for the acid-fast property of bacteria occurring in cases of cerebral abscess, gastric cancer, pulmonary abscess or even disease of the antro-tympanic cavity.

Dr. MILLIGAN said the contribution was a most valuable one, and that further study of it would reveal points of great practical value. As to whether valuable clinical information could be obtained by examining the discharge, he held that it could: and if the films were carefully prepared and stained, it was unnecessary in many cases to make cultivations. Inoculation experiments were difficult and tedious, and were not by any means always necessary. As otologists they required the practical application of the paper to their every-day work, and that application he thought was forthcoming. There were many cases which presented no difficulty from the clinical point of view: but there were others in which doubt arose as to which of two methods of treatment should be followed. In the latter cases cytological examination was of value. The question of tuberculosis in the ear had been raised many times at the Section. Very often it was difficult to diagnose the tuberculous nature of a case, and all admitted how difficult it was to find tubercle bacilli in the discharge. Sometimes portions of epithelial cells which possessed acid-fast properties became broken up, and looked and stained very much like tubercle bacilli. The presence of a large number of myelocytes, giant- and epithelioid-cells in a film was good evidence of the tuberculous nature of the case. Dr. Wingrave had shown that the finding of acid-fast squames in the discharge was valuable in the diagnosis of infective cholesteatoma. He could relate clinical cases to show the help he had received from such examination. One was a case of suppurative middle ear disease which presented anomalous symptoms, and which it was difficult to diagnose accurately; films from intertympanic washing out were made, and a large number of acid-fast squames were discovered, leading to the view that it was probably infective cholesteatoma. Operation disclosed the presence of a large cholesteatoma, which had eroded both backwards towards the cerebellum and upwards towards the cerebrum. The whole question was still more or less *sub judice*, as not very much work had as yet been done on it; but it was an inquiry well worth following out, as it led one forward in doubtful cases. Much depended on the way in which the films were made and the care with which the material was drawn from the ear. He had been working at the cytological aspect of the question for some time, and he was sure it was of definite and practical value in the diagnosis and treatment of cases.

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[1] "On the Relations of the Tubercle Bacillus to other Bacteria resistant to Acids and to Actinomyces," by Dr. Moeller (Belsig), *Trans. Brit. Cong. Tuberculosis*, 1901, iii., p. 485.

[2] "The Morphological and Physiological Variations of the *Bacillus tuberculosis* and its relations (*a*) to the other acid-fast bacilli; (*b*) to the ray fungus and other streptothriceæ," loc. cit., p. 494.

Dr. KELSON also thanked the author for his admirable paper. It contained no mention of diphtheria, but one often saw cases of obstinate discharge from the ear after that disease. He asked whether there were Klebs-Löffler bacilli found in those cases, or whether there was reason to think that contagion was spread in that way. He had met with a case in which, after a bad attack of scarlet fever in which only aural discharge remained, there was reason to think that the disease was transmitted to two families. He would be glad to hear more about cholesteatoma and allied conditions, especially as to whether there were any bacterial agency at work there. Sometimes such conditions interfered with healing after the radical and other operations.

Dr. GRANT said he thought there could scarcely be any question about the practical value of Dr. Wingrave's work, and he was sure Dr. Horne had the greatest sympathy with the author in the work he was carrying out. He wished that Dr. Wingrave had been able to follow out the clinical histories of all the cases which he had examined bacteriologically. Perhaps the failure to do so was partly due to the partial support given to the work by those working at the hospital, but such was very difficult in the immense crush of work there. The author's efforts tended to make otology much more scientific, and if the after-histories of cases were more constantly placed at his disposal he believed that a future paper based thereon would answer all criticisms. Even though the result of cytological examination was not so absolute as some expected, the great value of the method would be confirmed.

Dr. DAN MCKENZIE wished to draw attention to a point in the paper which, despite its novelty and importance, had not received mention from the previous speakers. He alluded to Dr. Wyatt Wingrave's discovery of spirochætæ and other mouth organisms in purulent discharges from the middle ear as well as in those of cerebral and cerebellar abscesses. If future investigation proved these organisms to be pathogenic—and certainly their presence in brain abscess was very suggestive—Dr. Wingrave's work on the subject would, on that single point alone, mark an advance. It seemed, indeed, as if one more disease was about to be added to the long list which they knew to be due to the presence of oral sepsis. He mentioned gastro-intestinal infections, pernicious anæmia, gastric ulcer, obscure forms of pyæmia, fœtid bronchitis, and follicular tonsillitis as having been ascribed to oral sepsis, and Dr. Wingrave's discovery naturally led to the supposition that, even if they did not originate the disease, mouth organisms at least helped to perpetuate suppuration in the middle ear. He desired to add his congratulations to those already expressed by the others.

The PRESIDENT expressed the thanks to the author, not only of the Otological Section, but of otologists everywhere. He did not profess to be sufficient of a microscopist to criticize the contribution, but he was sufficient of a clinician to appreciate the work which Dr. Wingrave had done.

Dr. WINGRAVE, in reply, thanked the various speakers for their remarks. He confessed to some extra enthusiasm in regard to the examination of aural discharges, which he had been carrying on for about fifteen years, though careful record was confined to the last 500 or 600 cases. He agreed with Dr. Horne as to the necessity of combining film staining with cultivation, for by cultivation alone many important bacteria might be missed. Examination was of no use unless done thoroughly. His plan of having three methods would, if intelligently carried out, suffice to identify to a considerable extent the chief morbid changes in progress. He first used the Gram stain to decide to what group the bacteria belonged. Then he used a stain for acid-fast bacilli and afterwards supplemented it by a process, according to the bacteria which he found. Patience and perseverance must be exercised, nor could familiarity with the histological details be quickly acquired. He would be only too pleased to classify the organisms into pathogenic and non-pathogenic, but there was much evidence that many bacteria enjoyed a twofold existence. In the throat, what were generally recognized as pathogenic organisms could be found passing a non-pathogenic existence; these included diphtheria bacilli, Hoffmann's bacillus, pneumococci, meningococci, &c., yet one could not consistently say that they passed a non-pathogenic existence there and pathogenic in the ear. Spirochætæ might be comparatively harmless in the throat, but they played an important rôle in the middle ear and in cerebral abscesses. With regard to acid-fast properties a contribution had been published by Professor Deycke[1] in which he described a neutral fat which had been separated from the leprosy and tubercle bacilli, and that that constituted its active principle, including its leucocytic influences. He had not been privileged to carry out any animal experiments as he did not possess a licence for the purpose. He agreed with Dr. Milligan that much clinical value could be attached to the examination he had described in a non-cellular discharge, because there it afforded an illustration of polymicrobic infections without any leucocytes or tympanic epithelium. In quiescent cholesteatoma there were no bacteria whatever. In a measure the information was often collateral, and he did not think anything could be more important than the discovery of what might be called throat bacteria in aural discharges, as it showed that the throat must be dealt with before it was reasonable to expect a chronic aural suppuration to be successfully treated. With regard to the differentiation of acid-fast squames, he had no doubt that even from a superficial examination ample information of a cholesteatoma could be derived, provided that the discharge was taken near its exit from the antro-tympanic cavity, since acid-fast squames were in abundance at the meatus. His paper only touched the fringe of the subject, but perhaps it would stimulate further investigation. There was no reason why an intelligent investigation of aural discharges should not be of equal value as that of urine and sputum. Unfortunately none of the present works on cytological pathology dealt with the matter. He agreed with Dr. Milligan as to the usefulness of removing discharge by means of the platinum loop. But when the discharge was plentiful, and it could not be examined the same day, a pipette was very useful if it was sealed up at once. In answer to Dr. Kelson he had not been able to say that the diphtheria bacillus was present in acute suppurations. It was found in old chronic cases and in acute exacerbations, while diphtheroid bacilli were frequently found in the throat.

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[1] *Brit. Med. Journ.*, 1908, i., p. 802.

Lateral Sinus Disease: Operation; Cure.

By W. S. Syme, M.D.

The patient, a boy aged 6, was admitted into the Glasgow Ear, Nose and Throat Hospital, suffering from a small painless swelling behind the right ear and an otorrhœa of four weeks duration. The tympanic membrane was destroyed in the posterior part. His temperature and pulse were normal and there were no symptoms to cause anxiety. The case was looked upon as one of slow caries of the mastoid antrum and cells. The radical mastoid operation was performed on November 13, 1906. The antral cavity and mastoid cells were found to be turned into one cavity filled with carious debris, the posterior wall was destroyed, and the lateral sinus was exposed to a large extent. The wall of the sinus was covered with grey, sloughy-looking granulations, which bled easily when touched, and which were not otherwise interfered with. The operation was concluded in the usual way. The cavity was packed, but the incision was left open in view of the condition of the sinus wall. A partial facial paralysis followed this operation. For three days his condition was satisfactory, but on the evening of the fourth day his temperature rose to 102° F. Thereafter he had rises of temperature with remissions, but no actual rigors. Looking to the unhealthy state of the sinus wall, I was inclined to look upon the condition as one of toxæmia rather than of actual septic thrombosis of the sinus. The cavity was therefore dressed daily with wet carbolic dressings. On November 24, as there was no improvement, I exposed the sinus more fully posteriorly, where the wall appeared healthy, and downwards towards the bulb, in which direction the grey and unhealthy appearance of the sinus still persisted. On slitting open the vessel it was found that for $\frac{3}{4}$ in. in its long axis and for half the circumference the wall was thickened, and to the inner surface a firm, dark clot was adhering. On removing the packing between the bone and sinus, copious hæmorrhage occurred, but on controlling the upper part, the flow from the lower part was only of moderate amount and was easily checked. This, I considered, pointed to a thrombus lower down, partially obliterating the lumen. The sinus was packed. As after waiting thirty-six hours the temperature still pointed to septic absorption, I ligatured the internal jugular, which, however, was not thrombosed at the part exposed. The incision in the neck was sutured. The result of this procedure was an improvement in the patient's condition. The wound in the neck closed in a few days, but at the end of a week broke down and discharged a fair amount of pus for five weeks, when it slowly healed. The mastoid cavity took a long time to fill up, and the boy left the hospital after a stay of twelve weeks. At the present time the ear is quite dry.

There are one or two points of interest in this case to which I should like to direct attention. The mastoid disease developed in a most insidious manner without pain, and it was only the appearance of the small swelling which excited suspicion. Yet by this time the bone had been extensively excavated, and looking to the condition of the sinus at this stage, he was evidently on the eve of a serious septic sinus thrombosis. Probably the course of events in the sinus was the following: The inflammation of the wall led to a slowly formed mural thrombosis at the affected part. For a time the actual ingress of organisms was resisted, but either from weakening of the wall or from the exposure of the granulations at the time of the mastoid operation, this resistance was overcome, and a secondary and infected thrombosis occurred in the lower part of the jugular bulb. It is worth noting that the early changes in the sinus occurred without fever, and that there was an absence of rigors even later, though this is not unusual in children. The internal jugular was tied only after the operation on the sinus seemed to have failed to effect improvement. I say "seemed," because it is open to anyone to contend that sufficient time did not elapse between the two procedures.

The boy remained in a somewhat stupefied state for several days after the ligature of the jugular. The discharge of pus through the wound in the neck was, it seemed to me, the result of the breaking down of the clot in the sinus and upper part of the jugular, which found in this way a means of escape.

I regret that a bacteriological examination of the discharge from the ear was not obtained. Whatever the organism or organisms present, I am inclined to think we had to deal with a mild infective agent, and that this conduced to the successful issue.

DISCUSSION.

Mr. A. **CHEATLE** asked why the radical operation was done. There was evidently acute suppuration, and it seemed a pity to destroy the middle ear under those circumstances.

Dr. **FITZGERALD POWELL** said that the sinus was exposed at the operation, and asked why it was not thoroughly examined at the time.

A Case of Bezold's Mastoid Empyema which discharged into the Pharynx.

By W. S. SYME, M.D.

THE history of this case, a man aged 52, is that a purulent discharge from his right ear, from which he had suffered for eleven weeks, had ceased three weeks before I saw him, but after its cessation a painful swelling appeared behind the ear and increased in size for two weeks, when suddenly he spat up a large quantity of pus, and pus also flowed from his nostrils. The swelling became smaller. Since then, however, it had again increased, and on examination it was not difficult to diagnose a Bezold's mastoid empyema. On looking into the throat pus could be seen trickling down the right side of the pharynx, but a detailed view of the naso-pharynx could not be obtained. Mentally the man was dull and lethargic. The walls of the meatus were so swollen that the condition of the membrane could not be determined. On operation the whole mastoid process was found to be excavated and the inner wall destroyed. The antrum was not in communication with the diseased part, and was therefore not opened into. The abscess in the soft parts extended deeply beneath the angle of the jaw, but no special effort was made to find a way into the pharynx for fear of getting subsequent trouble from a fistulous opening. The ultimate course of the case was in every way satisfactory.

Injury to the Pharyngeal Portion of the Eustachian Tube from Operative Procedures.

By W. S. Syme, M.D.

The patient, a woman, stated that some months before she was sent to me she had been operated on on two occasions for nasal catarrh. After the second operation she had become quite deaf in the right ear, although previously her hearing in both ears was very good. When I saw her the tests were—Watch: right ear 0, left $\frac{30}{40}$; Rinné: right ear −, left +; Weber: right. The more detailed tests showed nothing of interest. Both membranes, but especially the right, showed loss of translucency with adhesions and indrawing. In the naso-pharynx there was much purulent discharge with crusts. After removing these it was seen that the right Eustachian prominence was absent, the left was normal in size and position. There was also destruction of the posterior part of the nasal septum. Catheterization was performed with difficulty on the right side, and only a very slight improvement in hearing resulted. On the right side of the pharynx, just above the level of the palate, was a rounded protuberance about the size of a large pea. This was firm to the touch and I took it to be the Eustachian cartilage torn from its attachment and drawn into that position by the action of the levator palati or dilator tubæ. As far as I could gather forceps had been introduced through the nostril and used without either knowledge of the anatomy or regard for the structures. A secondary adhesive inflammatory condition had arisen in the tympanum, and this, combined with the constriction of the tube, had led to disastrous consequences as regards the hearing.

DISCUSSION.

Dr. Milligan said he had previously shown photographs of a case of bilateral facial paralysis due originally to Bezold's mastoid disease. The patient had a mastoid empyema, and sudden symptoms of post-nasal obstruction supervened, followed by the evacuation of a considerable amount of pus and debris from the nose and throat. When syringing from the external wound fluid came straight through into his pharynx. The other ear became infected as a result of the post-nasal suppurative condition, and facial paralysis supervened.

The PRESIDENT, in connection with the third case, asked what was the appearance of the membrane and the result of the tuning-fork test. Also what evidence was there, beyond the patient's statement, that the hearing was good before?

Dr. SYME replied that the reason he did the radical operation, although there was an acute history, was that there was so much destruction of mastoid, involving the posterior wall of the meatus, he did not think any less would be efficacious. The sinus was not examined because the wall was covered with granulations, and he did not think it wise to interfere with those under such circumstances unless one was certain there was suppuration in the sinus at the time. At the summer meeting of the British Medical Association he described a case where interference with granulations in the tympanic roof led to infection of the meninges. With reference to Dr. McBride's question, he regretted that, under a misapprehension, he did not put more particulars into the published notes. The patient said that before coming to him she had been twice operated upon by a medical man, and that after the second operation she became quite deaf in the right ear. With the watch the hearing in the right ear was 0, and in the left ear $\frac{30}{40}$. The more detailed tests showed nothing of interest. There was loss of translucency and indrawing of both membranes and absence of the right Eustachian prominence. There was a swelling above the palate on the right side, which was firm to the touch, and gave the impression that the Eustachian cushion was pulled down. He had only the word of the doctor who saw her previous to the first operation that she was all right before, but this was not denied by the medical man who operated.

Case of Unilateral Hysterical Nerve Deafness of Sudden Onset, with Hemianæsthesia and other Allied Stigmata.

By J. DUNDAS GRANT, M.D.

WOMAN, aged 27, first seen February 12, 1908. Complained of deafness in right ear. Onset sudden, with pain in head. Duration four months. Comparative hemianæsthesia right. No vertigo or nystagmus on rotation. Marked narrowing of field of vision. Galton's whistle only heard at mark 11. Tuning-fork (C^1, 256 D.V.s), when first seen, not heard either at the meatus or the mastoid; on the vertex it was heard only in the opposite ear; Rinné's test could not be reliably taken. Her symptoms have since then somewhat diminished.

DISCUSSION.

Dr. PURVES STEWART said that there were certain other stigmata present, namely, concentric diminution of field of vision, impairment of taste, and diminished common sensibility—all on the same side as the nerve deafness. Those, combined with hemianæsthesia, were pathognomonic of hysteria. The patient had sudden paroxysms during which she heard quite well. It would be interesting to hear from Dr. Grant whether on such occasions the taste and vision returned to the normal. All the phenomena he had mentioned were right-sided. In the majority of hysterical patients functional phenomena were on the left side, with the exception of left-handed patients. Was this patient left-handed?

Dr. DAN MCKENZIE said he did not know whether attention had been drawn to the occurrence or absence of nystagmus and staggering in hysterical nerve deafness. He was responsible for the tests made on this patient. These were conducted in the presence of the students and visitors at the Central London Throat and Ear Hospital, and consisted of the usual rotation tests, the patient being seated in a chair slung from the ceiling by two ropes. After rotation in either direction there was not the slightest sign of nystagmus on extreme lateral deviation of the eyes; in like manner staggering was absent after rotation, and the patient when questioned said she felt no giddiness.

Mr. YEARSLEY asked whether Dr. Grant used for the tests the ordinary Galton's whistle or the Edelmann whistle. In the latter case it was more accurate to record the number of vibrations heard by the patient, which could be done by means of the table supplied with each instrument. He was sure the ordinary Galton's whistle was not always trustworthy, and he would like to hear the opinion of some of the Fellows present as to the accuracy of Edelmann's whistle also.

The PRESIDENT said that some time ago he went into the literature of the subject of hysterical deafness in connection with some cases he had seen, where deafness was the only symptom. It seemed to him that where there were no other stigmata the true diagnosis could only be certain after the case was cured. Eighteen months ago he published the case of a girl who had suddenly become deaf some weeks before. Though aged 19 she was still at school, where there had been an epidemic of mumps. The first day his note was "deafness hysterical or due to mumps." He put her on tonic and other treatment. Some months afterwards the history was that she had suddenly had "a rushing in her head" and her hearing recovered. It was pure nerve deafness; there was no middle ear involvement. He hoped Dr. Grant and Dr. Purves Stewart would touch on the question of diagnosis.

Dr. HASLAM asked what the constitutional condition of the patient was. Had she any neurasthenia?

Dr. GRANT, in reply, said hysterical nerve deafness took different forms. When unilateral it was probably part of the hemianæsthesia present in so many cases. When it was bilateral the diagnosis was not so easy. He had published a case in which he made the diagnosis on the strength of the hearing being equally low for the notes in all parts of the scale, not chiefly for the highest. That patient learned lip-reading without knowing it. She eventually recovered her hearing as a result of an illness, probably by producing a counter-irritation of the system. Deafness for all tones equally was more likely to be central than where hearing was lost only or mainly for high-pitched tones. The present patient's hearing, however, did not quite bear that out, as it was markedly diminished for high-pitched tones: she could not hear the Galton whistle at all. He still used the old-fashioned Galton whistle, but he endeavoured to get one graduated exactly in millimetres. He would be glad to hear whether the improved whistle was much better. There were, however, many other disturbing factors of greater importance than an imperfection in the whistle. The constitutional condition was, in the case exhibited, extremely marked.

Dr. PURVES STEWART further said he agreed with the President as to the difficulty of diagnosing hysteria from one symptom alone, but mono-symptomatic hysteria was the rarest of all and occurred chiefly in children. In most cases of hysteria several stigmata could be discovered if carefully searched for. The determination of whether labyrinthine deafness was functional or organic was largely helped by the observations of Bárány in regard to the production of nystagmus by the application of intense cold or heat. Cold water caused contralateral nystagmus—sometimes lateral, sometimes rotatory. He had experimented on several patients in hospital, and they all complained of severe vertigo and developed nystagmus. He also made experiments on himself and developed vertigo and rotatory nystagmus. In functional disease that nystagmus should still persist, but in organic disease of the auditory nerve vestibular vertigo should be reduced. The results obtained by the revolving chair sounded contradictory, because one would expect nystagmus to be produced in healthy persons by a rapidly-revolving chair.

The PRESIDENT, in reference to Dr. Purves Stewart's remarks, said that Bárány assumed that nystagmus due to syringing was vestibular. He (the President) said he understood it happened whether the membrane had been perforated or not. He asked how far the symptom might be merely a reflex through the sensory nerves. The labyrinth might be, and probably was, a factor; but were we sufficiently certain of the *modus operandi* to enable us to attach great value to the method as a means of examining the labyrinth?

Dr. PURVES STEWART replied that cold at the side of the head did not produce it, it was only produced when the cold water reached the tympanum. In his own case it did not develop until he had a horrible feeling of coldness at the tympanum. By a process of exclusion there was no other factor to produce the symptom than coldness.

Dr. MILLIGAN asked whether there might not have been some reflex from the tympanum or from the auditory meatus. It did not follow that the cold fluid touched the labyrinth at all in the case of an intact membrane. There was a definite column of air between the membrane and the labyrinth.

Case of Unilateral Nerve Deafness in an elderly Man.

By J. DUNDAS GRANT, M.D.

MAN, aged 70, complained of deafness in right ear. Duration one year, after nervous breakdown. Sudden onset. Frequent attacks of vertigo. Double vision on looking upwards and to the right. Unsteadiness diminished by closure of the eyes. Probably two separate lesions. History of former specific infection. Scar on the right half of hard palate. Comparative hemianæsthesia, which has since become much less marked. Galton's whistle not heard at all on the right side, but at mark 1·6 on the left. The tuning-fork (C^1, 256 D.V.s) not heard at the meatus, and diminished to the extent of fifteen seconds on the mastoid; on the vertex only heard in the good ear; Rinné's test uncertain.

DISCUSSION.

Dr. PURVES STEWART said there was evidently an organic lesion in the region of the right auditory nerve. The patient had organic deafness and weakness of the right sixth nerve. The coexistence of another cranial nerve palsy in the trouble excluded functional disease. The sixth nerve had but little localizing value, and could not be used for diagnosis. Pain in the right orbit and the absence of signs of implication of any of the long tracts of the brain stem, either sensory or motor, pointed to the affection being superficial, at the base of the brain. There was not sufficient evidence to show its nature. The patient had been in the Navy and had been treated with mercury and iodide of potassium. He suggested lumbar puncture and the examination of the cerebrospinal fluid. If the disease was specific that fluid would show lymphocytosis.

Mr. YEARSLEY asked whether the case might possibly be one of the rare forms of auditory tumour described by F. Hartmann.

Dr. MILLIGAN asked whether the sudden onset did not rather suggest some vascular change. Was it syphilitic endarteritis, with sudden occlusion of a vessel?

Dr. PURVES STEWART said he agreed with Dr. Milligan that syphilitic disease included endarteritis, and possibly a sudden vascular thrombosis in the region of the ear. Lymphocytosis of cerebrospinal fluid would corroborate the diagnosis. When the patient shut his eyes the vertigo disappeared; it was therefore ocular in origin, not central.

Specimens of Necrosis of the Labyrinth.

By P. Macleod Yearsley, F.R.C.S.

K. B., aged 22, was sent to me from the Heart Hospital by Dr. C. C. Gibbes. She gave the following history: She had suffered from discharge from both ears, on and off, since measles in childhood. That from the right ear had been very much worse since last July, was very offensive and stained with blood. About September 26 last she had an attack which she called "inflammation of the brain"; she was attending a meeting, when she fell forward from her chair and was carried home unconscious. She was very ill from that date until December, but I have been so far unable to obtain any details of her illness, save that it was accompanied by a good deal of vertigo and tinnitus, which she likened to "being in a railway station." I can obtain no details of her giddiness, save that early in March she had a sudden attack, when she fell to the right.

When seen on April 30 the girl was obviously ill; the left ear was discharging very slightly. The lower part of the membrane was destroyed and the handle of the malleus hung free. The right meatus was full of very foul pus stained with blood, and on cleansing, large polypoid masses were seen blocking the inner end of the canal. She appeared to be quite deaf on the right side, but heard well on the left. She was, however, too ill to be tested. There was no nystagmus. She was at once admitted and prepared for operation. The temperature on admission was 98° F., pulse 100, of fair quality. On the morning of Thursday, temperature and pulse were 97° F. and 80.

On April 30 she was anæsthetized, and the radical mastoid performed. On reaching the antrum, very offensive curdy pus appeared, and, as the opening was being enlarged, escaped as if it were being pumped out. On removing the bridge, the antrum and middle ear were cleared of much granulation tissue, and it was then found that the inner antral and tympanic wall was occupied by a bed of granulation tissue, across which the facial nerve could be seen lying exposed. Loose sequestra were felt and removed, two large and two small. The two former comprise a part of the vestibule, with the openings of the semicircular canals, and a much eroded portion of the promontory. With the latter was a small piece of the cochlea, showing part of the lamina spiralis ossea.

The condition of the patient was such as to make it inadvisable to complete the operation, and the wound was therefore lightly packed from the incision. She rallied well, and is now progressing favourably, but slight facial paralysis, chiefly affecting the eye, is present.

Case of Chronic Eczema of both Auricles and Meatuses with unusual degree of Hyperplasia in a Woman aged 43.

By E. Furniss Potter, M.D.

The condition is of three to four years duration, and is stated to have commenced with irritation behind the ears (mastoid region). Patient thinks that prior to this she had discharge from both ears, but the evidence as to this is indefinite. No sign of otorrhœa at present, lumen of meatuses so much obstructed that view of deeper parts impossible. Both auricles are irregularly thickened, and swellings in conchæ which overlap the meatuses give impression at first glance of a large polypus protruding from the meatus.

DISCUSSION.

Dr. FitzGerald Powell said he thought the conditions surrounding the external meatus were keloid in character, following chronic eczema; he thought it would be well to have a portion removed for microscopical examination.

Dr. Dan McKenzie thought the swelling was due to œdematous infiltration of the subcutaneous tissues of the auricle. The perichondrium of the auricle was closely adherent to the cartilage, and Dr. Wingrave had shown that the blood-vessels lay in this layer. Consequently the circulation was liable to be blocked, and the œdema became persistent, a by no means uncommon event in chronic eczema of the auricle. On the left side in this case could be seen the first stage of the complaint and on the right side the same condition more advanced. He suggested that the state of matters in this case was merely the ordinary eczematous exudation somewhat exaggerated.

Mr. Yearsley said he saw a case in which one ear was affected very much like the present one. It cleared up under treatment, and seemed to have been due simply to chronic eczema which had been neglected. In that case the eczema had existed, on and off, for five years, and there was a mass of swelling in the concha. His assistant had brought it to him as a possible case of epithelioma.

Dr. KELSON said Dr. Potter told him he did not know quite what to call the case, but inclined to the term condylomata, but without suggesting syphilis. It might have been due to an irritating discharge affecting the subcutaneous tissue.

Case of Complete Occlusion of the Posterior Nares with Flattening of the Nose externally and almost complete Absence of the Nasal Septum.

By HUNTER TOD, F.R.C.S.

THE patient, a male child aged 1, was born healthy. Snuffling from the nose after first month. Had a rash on the body for a few weeks at the age of 6 months. The mother has been ill ever since this child was born.

On examination under a general anæsthetic, the post-nasal space was felt by the finger to form a smooth round surface with complete absence of the posterior choanæ. Anterior rhinoscopy showed complete absence of the cartilaginous and most of the bony septum. On passing a probe along the floor of the nose, whilst the finger of the other hand was kept in the post-nasal space, the end of the probe could be felt to impinge against what appeared to be fibrous tissue. The complete occlusion was demonstrated by injecting milk into the nose and carefully observing whether it trickled down from the post-nasal space into the pharynx; this did not take place.

The diagnosis is, provisionally, that of congenital syphilis. Opinions are invited with regard to treatment. There is no apparent difficulty in forming a passage through the nose, but the question is how to retain the opening made. The best method would probably be retention of rubber tubing passed through the nose and brought out through the mouth, and kept permanently in position for several months. Is this method of treatment practicable?

DISCUSSION.

The PRESIDENT said that, speaking without a minute examination, he inclined to the view that it was specific.

Dr. GRANT agreed with the President, but did not think the occlusion was now specific, but of a fibrous character, such as might occur from severe suppurative inflammation of the posterior choana, the remains of syphilis. If it were in an adult one would puncture through the nose and keep a tube in for a considerable time, perhaps even always, but in such a young child, with that history, he did not recommend it. Eventually it would, perhaps, be necessary to remove the posterior portion of the nasal septum.

Mr. TOD, in reply, said that his chief object in bringing the case was in reference to treatment. He did not see how a tube could be retained in position unless it was brought out through the mouth and tied, and he thought this would be impracticable. He would therefore leave the case for the present.

Otological Section.[1]

June 27, 1908.

Dr. PETER McBRIDE, President of the Section, in the Chair.

Middle Ear Suppuration; Phlebitis of Lateral Sinus; Cerebellar Abscess.

By P. McBRIDE, M.D.

PATIENT, male, aged 23. First seen October 14, 1907, with Dr. George Hunter, who had treated him between the ages of 1 and 7 for right-sided ear discharge, and had then removed pieces of lower jaw and temporal bone. The left ear had discharged for two or three years and had been treated by syringing. Pain came on four days before his visit to me. On examination the auricle was projecting, but there was no great tenderness on pressure, excepting in front of the tragus. Pulse 84; temperature normal. A mastoid operation was recommended; but two days later a good deal of offensive pus came away, and the patient absolutely refused interference as pain had ceased. The meatus contained granulations and exposed bone was felt. The right ear was deaf and the left almost entirely so. Owing to the patient's attitude no further examination was made at this time, and the case was not seen again until January 14, 1908. The history then showed absence of pain, but giddiness with rotation referred to surrounding objects for the previous fortnight. Vomiting had occurred a week earlier, and there was nystagmus on looking to the right. The meatus was contracted, but there was no mastoid tenderness. The tuning-fork and other tests pointed to involvement of the middle ear rather than the labyrinth. The patient now consented to operation.

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[1] First Provincial Meeting, held at the Royal Infirmary, Edinburgh.

I performed a radical mastoid operation on January 16, closing the posterior wound and using Brühl's plastic. After the operation the temperature showed slight evening rises for five days (up to 101° F.), was rather subnormal on January 22 and 23; on the evening of the 24th it was 99° F., on the 25th 100° F., on the 26th 103° F., and on the 27th 104° F. There were no rigors, nor was there any leucocytosis. After this it fell, but four-hourly charts showed marked fluctuations. There were no changes in the discs. It was rather difficult to exclude influenza. The pulse rarely exceeded 100. On February 5 I exposed the lateral sinus and turned out a large clot, getting a flow of blood both from above and below. The jugular vein was not ligatured. On the following day the temperature again rose to 104° F., but on February 7 the highest point reached was 102·4° F. Next day it did not exceed 100° F. After this it rarely rose above 99° F., and on the average was subnormal. Pulse from 78 to 90.

The patient during this period seemed rather inclined to sleep, and on February 16 there were vomiting and stupor. On February 17 these were more marked. There was nystagmus on looking to the left. Dr. Hunter and I concluded that another operation was required, but that as I had already operated twice it would be better to give the relatives an opportunity of calling in a surgeon if they desired it. Of this they availed themselves, and it was suggested that Mr. Dowden should be summoned, and, if he agreed with our opinion, should explore the cerebellum, and, failing pus there, the temporo-sphenoidal lobe. Mr. Dowden accordingly operated on those lines on February 17, but exploratory puncture failed to discover pus in either situation. Pressure, however, was relieved and the patient improved, the pulse rising from 78 to 110 and the stupor passing off. After a few days it was seen that pus seemed to come up from below along the track of the sinus, and it was further observed that as this pus increased the patient was better, but when it diminished he became drowsy. The pulse again became slower, and on February 28 stupor again set in. A further operation was therefore suggested, and on March 1 the already large opening in the skull was enlarged downwards. In spite of suction and probing, however, we could not find the track of the pus. This operation was again followed by relief, and on March 7 the abscess cavity, containing about half an ounce of pus, in the lateral lobe of the cerebellum, was found, evacuated, and drained by Mr. Dowden. Since that time the patient has made an uninterrupted recovery.

Patient operated upon for Sigmoid Sinus Thrombosis.

By A. Logan Turner, M.D.

E. D., aged 17, admitted under the care of Dr. G. A. Gibson with high temperature and pains in the back and shoulders, her condition suggesting influenza. She gave a history of having had a boil in the right external auditory meatus, which had been incised two or three days before admission. On day following admission she had a severe rigor, and this was repeated at intervals. She did not complain of any localizing symptoms, but the history of the boil caused attention to be drawn to the right ear. Ear examined by Dr. Turner on December 7. No mastoid tenderness; no swelling; right tympanic membrane normal. Lumbar puncture negative; leucocytosis 16,800. Rigors continued. December 9: Ear again examined; mastoid tenderness now present; no œdema; slight bulging of posterior superior quadrant of membrane; hearing defective. Discs normal; leucocytosis 16,800. Operation on same day. Paracentesis of tympanic membrane; drop of pus evacuated; pure culture of *Staphylococcus pyogenes aureus*; mastoid cells contained no pus; bone not specially inflamed; sigmoid sinus pressed backwards with probe in order to gain access to antrum; a little pus escaped from antrum. Sinus wall exposed for 1½ in.; wall did not look unhealthy. Sinus incised; dark clot filled lumen; right internal jugular vein ligatured. Large clot removed from sinus; pure culture of *Staphylococcus pyogenes aureus* obtained from it. On account of recent character of case the complete mastoid operation was not performed. This, however, was found necessary some months later as a fistulous opening discharging pus persisted over the mastoid, and discharge continued from the middle ear.

Patient after Operation for Left Temporo-sphenoidal Abscess occurring in the course of a recent Middle Ear Suppuration.

By A. Logan Turner, M.D.

A. O., aged 36, had acute inflammation of the left middle ear in July, 1907. There was no history of any previous ear affection. On October 13, 1907, patient was examined; temperature 101° F., pulse 80.

Great pain in left ear and on left side of head; tongue dry and brown; mastoid tender; tympanic membrane bulging above and behind; mucopurulent discharge; she had been vomiting; could answer all questions when spoken to. Mastoid operation performed. Marked improvement in patient's condition. October 20: Temperature fell to 97° F. and 96° F., pulse to 54 and 44; headache again troubled her; becoming drowsy and dazed; no changes in either fundus; no abnormal pupil phenomena; no ocular paralysis; loss of the power of naming objects. Leucocytosis 13,000. Polymorphonuclear cells 88 per cent. Large abscess evacuated from left temporo-sphenoidal lobe through roof of antrum. *Streptococcus pyogenes* and *Streptococcus pyogenes aureus* in brain abscess.

Macroscopic and Microscopic Preparations from a Case of Right Temporo-sphenoidal Abscess and Left Sigmoid Sinus Thrombosis.

By A. LOGAN TURNER, M.D., and HENRY WADE, F.R.C.S.Ed.

R. M., AGED 19, had suffered for a number of years from bilateral middle ear suppuration. In 1900 a right temporo-sphenoidal abscess was successfully drained by Mr. J. M. Cotterill. In March, 1907, Dr. Turner performed a radical mastoid operation upon the left side, ligatured the left internal jugular vein, and removed a thrombus from the sigmoid sinus. A pure culture of *proteus vulgaris* was obtained both from the mastoid abscess and from the clot in the sinus. The clot in the jugular vein yielded no growth.

The patient was readmitted in October, 1907, showing signs of considerable emaciation; inability to recognize his friends; he was unable to walk without support, having a marked tendency to fall to the right. There was nystagmus of both eyes when rotated to the right, and weakness of the left external rectus muscle. There was considerable tremor of the left arm in voluntary movement; marked exaggeration of both knee-jerks and ankle-clonus. No Babinski. Dynamometer, right hand 15, left hand 25. Deafness was so marked that it was impossible to carry out satisfactory tuning-fork tests. There was a distinct tender area on pressure over the anterior and upper part of the left side of the cerebellum. Increasing drowsiness; temperature 97° F. to 96° F., pulse 64. Leucocytosis 17,600. Polymorphonuclear cells 80 per cent. After consultation with Dr. Edwin Bramwell the left cerebellar lobe was explored, but no abscess was found. Death on the fourth day following the operation. No abscess found post mortem.

A Case of Infection of the Lateral Sinus; Pneumonia and Pericarditis; Ligature of the Jugular Vein; Complete Recovery.

By W. Permewan, M.D.

Boy, aged 10, admitted with history of acute ear disease of five days duration. Temperature had varied from normal to 105° F., but, according to the doctor, no rigors. On admission temperature 103° F., tenderness but no swelling behind mastoid. The antrum and mastoid cells were opened, and contained pus; mastoid cleared out. Next day rigor; temperature 105·5° F. Lateral sinus explored; sinus thrombosed and contained pus. Sinus wall freely opened and thrombus cleared out above and below till bleeding occurred, then plugged. Internal jugular vein then tied, and 1 in. excised. Next day pericardial friction heard, and a dull patch on the right lung with pneumonic breath sounds. Antistreptococcic serum administered and repeated several times. Recovery slow but complete; patient remained well since.

DISCUSSION.

Mr. A. L. Whitehead asked Dr. McBride whether in this case the cerebellum was explored through a separate opening posterior to the lateral sinus or if the mastoid opening was followed backwards so as to expose the cerebellum. With regard to Dr. Logan Turner's cases, were the leucocyte counts continued after the operations until convalescence, and, if so, what were the results? In the cases of sigmoid sinus thrombosis, was the sinus exposed until a healthy portion was reached before the wall was incised? If this were done, there was less risk of pushing back a portion of infected clot by the gauze plug used to arrest the hæmorrhage, and the sinus could be more deliberately laid open and the inner wall inspected.

Dr. Thomas Barr asked, with regard to Dr. McBride's case, how the abscess was ultimately found in the cerebellum—whether it was reached by operation from the posterior of the pars petrosa in front of the cerebellum. There was another interesting point in connection with that case, viz., the symptom of nystagmus. He would like to know if this symptom became more marked as the disease advanced, or the contrary. He noticed that at first the nystagmus was on the right side and later on the left alone. Neumann and Bárány alleged that one could differentiate cerebellar suppuration from labyrinthine by the presence and behaviour of nystagmus. In the former the nystagmus, slight at first, became worse and worse as the disease advanced, whereas, in the case of labyrinthine suppuration, the nystagmus was more pronounced at first but gradually became less marked, till it disappeared. He asked whether the former course was noticed in this case. The non-existence of rigors in the case was noteworthy. Of course, the most striking feature of sinus involvement was repeated rigors, with violent fluctuations of temperature. This case confirmed what he had occasionally seen, that rigors might be absent, the sinus condition being indicated simply by marked fluctuations of temperature. These exceptional cases were a warning that they must not wait for rigors before operating.

Dr. MILLIGAN said it would be interesting to hear from the exhibitors of these various cases their reasons for tying the jugular vein or for leaving it alone. This point raised the question as to the class of case in which the jugular vein should be tied, and those cases in which it was sufficient merely to clear out the clot in the lateral sinus and leave the vein unligatured. It had always seemed to him that in those cases where there were no definite evidences of phlebitis extending down the neck, as indicated by tenderness, enlargement of glands, &c., one could obtain very good results by simply clearing out the clot and leaving the vein alone. In many cases, after opening the sinus no macroscopic evidence of infection of its coats was evident. On the other hand, where there was evidence that the coats were infected, and that the process was extending downwards towards the neck, then, he thought, there could be no doubt that the internal jugular vein should be tied. He raised this point because he noticed in Dr. Logan Turner's first case that he said: "Sinus wall exposed for 1½ in.; wall did not look unhealthy. Sinus incised: dark clot filled lumen; right internal jugular vein was ligatured." He thought one might reasonably ask Dr. Logan Turner why he did ligature the jugular vein in this particular case.

Mr. BALLANCE said it seemed to him that much was gained by modern methods of examination. He referred to the examination of the blood and of the cerebrospinal fluid, and the discovery of the organism producing the suppuration. Recently he had a very severe case of septicæmia, with symptoms also of meningitis, and in this case the vaccine treatment appeared to be most valuable. With regard to the question of ligature of the jugular vein in lateral sinus pyæmia or septicæmia, he thought the decision must depend upon whether the infection was a local or a spreading one.

Mr. HUGH E. JONES said he had not very much to add to what had been already said, but he would like to ask for information and help. In Dr. McBride's case of cerebellar abscess there was no mention of hernia; but as he then had a case of cerebellar abscess in which he had to plug the sinus, owing to tearing of its wall, which was necrotic and adherent to bone, during the necessary removal of bone, and in which a very large hernia formed, he would like to know whether, in the experience of those present, the plugging of the sinus, either by pathological processes or by operation, encouraged the formation of a hernia after the evacuation of the cerebellar abscess. He would also like to have some advice in regard to the treatment of hernia. In the case mentioned, everything had been tried except removal of the hernia: enlargement of the opening in bone and dura, the making of a counter opening, and search for a second abscess, painting the hernia with formalin and glycerine, &c. In regard to the question of tying the internal jugular vein, there was one thing that struck him in Dr. Logan Turner's notes. He said that no micro-organisms were found in the clot from the jugular vein. Many surgeons claimed to have found pathogenic organisms, not only in the clot but in the apparently healthy walls of the vein, very low down, in a large proportion of their cases (notably McKernon and Richards, of New York), and consequently practised the excision of the *whole* vein. It had always seemed to him that this radical proceeding was unnecessary, even where the upper part of the vein was filled with breaking down clot. It seemed sufficient to place the ligature below the clot and to excise the portion of vein above it, or to bring the upper part of the vein out of the wound and drain through it: but one must bear in mind that the results published by these gentlemen were very remarkable, and were possibly accounted for by their more radical method of operating.

Dr. KERR LOVE said that personally he had received in his practice almost no help from the study of the bacteriology of intracranial conditions: he had nearly always been able to make up his mind as to what course he should follow with regard to treatment, apart altogether from bacteriology. There was a case recently in which one of his colleagues operated and failed to find the antrum. A year later he (Dr. Love) operated upon the same case and was able to find the antrum, and a meningitis followed the operation. No treatment of any kind specially directed to the specific affection was adopted, and the girl made an excellent recovery. The diagnosis of meningitis was confirmed by an examination of the cerebrospinal fluid. Certainly, in these difficult cases any practical help given by an examination of the cerebrospinal fluid would be welcomed by aural surgeons, but in the meantime he felt that a careful study of the clinical symptoms gave as clear a guide as any study of the bacteriology of the case.

Mr. SYDNEY SCOTT asked Dr. Logan Turner and Mr. Wade, with reference to their case, what was the cause of death. It appeared to have been a very interesting case that Mr. Cotterill had operated upon some seven years ago—successfully draining a right temporo-sphenoidal abscess—and he saw that Dr. Turner operated on the left sigmoid sinus early last year. The symptoms with which the patient was readmitted in the autumn suggested to Mr. Scott that he was suffering from intralabyrinthine disease on the left side. As no cause for death was mentioned, and there appeared to have been increased intracranial pressure, he would also like to ask what was the condition of the ventricles of the brain after death, and whether they were distended with fluid or whether they were normal.

Dr. McBride, in reply, said that with regard to his case and the questions that had been asked, he would answer as shortly as possible. He might say that the cerebellum was explored simply by an opening made by extending that by which the sinus was exposed. The abscess was eventually found by Mr. Dowden. Pus was seen welling up along the lateral sinus from below, and the fourth operation was performed with a view to tracing it to its source. Eventually Mr. Dowden put a plug over the lower part, and then found a small orifice in the dura, which he enlarged. With regard to the question of nystagmus, his idea as to Neumann's views was that he believed persistent nystagmus on looking towards the diseased side was suggestive of cerebellar abscess, while the opposite condition was more commonly found in labyrinthine suppuration. This question of nystagmus greatly interested him, but as time was rather short he would not enter into it further. He might add that in his case there was no hernia.

Dr. Logan Turner, in reply, said he would confine his remarks to the clinical side of the case, and ask Mr. Wade to reply as to the pathological condition. First of all, with regard to the leucocytosis, the work in connection with this was done by Dr. Darling, and he would have preferred him to reply. The point was that, as far as the diagnosis was concerned between sinus thrombosis and a localized brain abscess, a much higher leucocyte count was found in the sinus thrombosis cases than in the cases of localized brain abscess. The leucocyte count was of value in connection with the prognosis of the case after operation; that was to say, allowing three or four days to elapse, in order to get over the effect of the anæsthetic, which again raised the leucocytosis, one found that daily, or every other day, the leucocyte count showed a gradual reduction to the normal. On the other hand, when the patient was not doing well, it was generally found that there was again a very considerable increase in the number of leucocytes. The second point raised was the question of ligation of the internal jugular vein in cases of sigmoid sinus thrombosis. Having exposed the sinus and come to the conclusion, either before or after opening it, that there was a clot, one at once ligatured the jugular. His reason for doing so was simply this: that he wanted to place a barrier in the blood-stream before disturbing the septic foci which were present in the sinus. Moreover, one did not know how far the clot might have extended down, and by exposing the vein in the neck one was able to learn this. The third point, which had not been raised in the discussion, but which he wanted to refer to, was the diagnostic value of the symptom of the loss of the power of naming objects in lesions of the left temporo-sphenoidal lobe. He had now had six cases, three of left temporal lobe abscess, one of large subdural abscess on the left side, and one of very extensive left-sided extra-dural abscess, and in all of them there was a loss of the power of naming objects. If, in a case of suppuration of the left middle ear, there was suspected intracranial abscess, the absence of the above symptom would be in favour of the pus being situated in the cerebellum. In a case at present in the ward, the cerebellum was first successfully explored and an abscess found, because this symptom was absent.

Mr. HENRY WADE, in reply, said he must, of course, confine himself entirely to the question of the pathological findings in the case shown by Dr. Logan Turner and himself. As far as he was aware, only one point had been raised to which a reply was required, but, with the President's permission, he would like to refer briefly to the more important pathological features of this most interesting case. To take them up here in the order they were raised, we had a successful operation for right temporo-sphenoidal abscess a matter of seven years before death. Now, the interest in this connection was that the abscess cavity had been replaced by what might be called a cyst. They found the brain and its membranes adhering by cicatricial tissue to the trephine opening, and the abscess membrane now replaced by organized cicatricial tissue. This cyst passed downwards for quite a considerable distance into the temporo-sphenoidal lobe, and was seen to be separated by only a delicate layer of brain tissue from the lateral ventricle. The next point was that in March, 1907, he was operated upon and the left internal jugular vein ligatured. They now found, of course, the usual change: the vessel was completely thrombosed for some distance. But there were certain rather interesting points. The first was that at the site of the ligature one could make out that although it was, roughly, nine months from the operation to the ultimate death of the patient from another cause, there was present a fragment of the catgut used to ligature the vessel. The sigmoid sinus was also completely thrombosed, and the lateral sinus showed a similar change. Its lumen was, however, not completely obliterated, and was lined by organizing granulation tissue. The auditory nerves on both sides were incorporated in masses of new growth, having the structure of a highly cellular fibroma. The cause of death was found to be explained by the presence of a small abscess in the pons. Time, however, would not permit of more detailed reference to certain other points of interest in this case.

Dr. W. PERMEWAN, in reply, said that he had only one remark to make, and that was that, with regard to tying the jugular vein, his reason was chiefly an experimental one. He almost always found that tying the jugular vein produced a good result. It had been said that the vein should not be tied unless there was phlebitis spreading into the neck. That was impossible to decide accurately: the alleged symptoms—pain and tenderness—were of no certain import; and, having seen an invariable improvement after ligature, he believed the sound practice was to tie the vein in every case. The alleged dangers of doing so were, in his judgment, wholly imaginary.

Two Patients after the Mastoid Operation with Preservation of the Tympanic Membrane and Ossicles.

By A. Logan Turner, M.D.

(1) J. C., AGED 34, had discharge from the left ear since childhood. During the last few years had suffered from occasional attacks of severe pain in the ear; headaches. On examination mastoid tenderness; fœtid discharge; Shrapnell membrane perforation. Hearing: watch $\frac{2}{50}$ in.; raised whisper 15 ft.; voice 21 ft. Operation February 28, 1908. Bridge accidentally fractured and removed; no displacement of incus; cessation of discharge, pain, and headaches; healing of perforation. Hearing varies a little, but has been as good as before operation.

(2) J. P., aged 23. Discharge from left ear for twenty years following measles. Suffered occasionally from headaches; discharge resisted careful meatal treatment. Perforation in posterior segment of membrane; discharge fœtid. Cytology: pus-cells scanty: polymorphonuclear cells; large squames; a few myelocytes; streptococci. Hearing: watch 4 in.; whisper 4 ft.; low voice 18 ft. B.C. > A.C. Lateralized to left ear. Operation October, 1907. Bridge broken and removed; no displacement of incus. Cessation of discharge; healing of perforation. Hearing sometimes better than before operation.

An Unusual Sequel to the Radical Mastoid Operation.

By W. G. Porter, F.R.C.S.Ed.

The patient, a boy, aged 16, was under Dr. McBride's observation for eight years suffering from an intermittent attic suppuration (perforation in Shrapnell's membrane). Gradual narrowing of the meatus and attacks of pain during the last year necessitated operation (Dr. McBride, June 27, 1907), although the hearing was excellent—watch $\frac{16}{30}$, ordinary whisper 18 ft. The radical mastoid operation had to be performed because the greater part of the disease (cholesteatoma) lay in the attic and tympanum; the outer wall of the attic was thoroughly removed, and the spur well flattened. During healing abundant granulations formed (no packing was used after first week), and the cavity became so stenosed that a second operation was required (Dr. Porter, August 30, 1907); a distinct ledge of bone was then found at the site of the outer wall of the attic leading to a recess, in the retiring angle of which the bone was carious. This was all removed, and a large meatus was cut. There was again very marked contraction, but healing was complete. October 8: hearing, watch $\frac{8}{30}$, whisper 18 ft.

The case presents the following points of interest: (1) In spite of the good hearing the pathological condition present necessitated a complete mastoid operation; a modified operation was inadmissible if anything like all the disease were to be removed. (2) There was an unusual and rapid new formation of bone. Was this an attempt at regeneration of bone? (3) The excellent hearing that was retained would appear to show that the functional result depends more on the presence of a intact stapes than on the retention of the ossicular chain. (4) Packing in this case merely stimulated the growth of granulations.

Notes on Partial Grafting, with the aid of Local Anæsthesia, in the After-treatment of the Radical Mastoid Operation.

By J. STODDART BARR, M.B.

THE treatment by packing, which has been so long practised by operators, when carried out with scrupulous care by the operator himself, yields, on the whole, good results. There is no doubt, however, that the time and trouble required form a burden grievous to be borne, and in the case of hospital patients it cannot be borne by the surgeon himself, and usually has to be transferred to others. Naturally, therefore, the operator is eager for some other way less exacting upon his time and patience which would yield equally good results.

By a combination of limited packing and partial grafting, without general anæsthesia and without reopening the post-auricular wound, I have been able to considerably shorten the healing process, and dispense to a great extent with the troublesome daily dressings. My practice is to insert a graft through the widened meatus some time during the second week after the radical mastoid operation. The technique I adopt is as follows:—The graft is cut from the left forearm or thigh under local anæsthesia induced in the following manner: A warm, sterilized, normal saline solution, to which has been added Parke, Davis and Co.'s codrenin in the proportion of 1 ampulla (10 minims) to 6 drachms of the normal saline solution, is injected under the skin from which the graft is to be taken. An ordinary hypodermic syringe, with a needle at least 2 in. long, is required, and several syringefuls of the fluid are injected in various directions before withdrawing the needle.

In this way it is quite easy to anæsthetize 3 sq. in. to 4 sq. in. of skin sufficient to permit of the removal painlessly of a large Thiersch graft. The graft, which must be thin to transparency, is then manipulated over the end of a suitably bent glass tube connected at the other end by rubber tubing having a glass mouth-piece or a small rubber bag. The graft at the end of the tube (somewhat like a closed umbrella) is passed through a wide speculum to the inner wall of the tympanum, when by blowing air through the tube the graft is spread out over the inner surfaces, including the tympanic walls, the aditus, and the antrum. (This may be compared with the opening of the umbrella.) Or one graft may be used for the tympanum and a second for the antrum. Before introducing the grafts the surfaces must be most carefully dried, and after their introduction xeroform powder is blown in so as to cover the grafts with a fine layer; afterwards small gauze tampons are carefully inserted into all the recesses. In a week the gauze packing can be removed, followed by gentle syringing with saline solution, drying, and a fresh insufflation of xeroform. In the course of a few days after removal of the packing the spirit treatment may be commenced. By this modified grafting operation and the limited use of packing, there is little doubt that the healing process is materially shortened.

DISCUSSION.

Dr. SYME said, referring to Dr. Logan Turner's remark that he accidentally fractured the bone in one case, that when going round he asked him why accidentally—why he did not always do so. He thought it would be wise always to remove practically the whole posterior meatal wall, although, as Mr. Whitehead said, it might be well to start with the intention of preserving the bridge and so make one anxious not to disturb the incus, and then finally to remove the bridge. By so doing the condition of the tympanum was better seen and its treatment could be more carefully conducted.

Mr. A. L. WHITEHEAD thought that there might be some advantage in trying to preserve the bridge until the last stage of the operation, so as to avoid injury to the ossicles. It should, however, be removed at the final stage, otherwise an area would remain from which diseased tissue could not be thoroughly removed.

Dr. KERR LOVE said that in reference to the cases under review the first point to consider was whether it was wise to do all one could to save the ossicles in a case of middle ear suppuration. His usual advice in such cases was to operate radically except in the case of a private patient. In a hospital patient it was usually foolish to try to save the ossicles where the suppuration was chronic. In nearly all of those cases coming before them in hospital the destruction had been so extensive that the attempt to save the ossicles would result in the radical operation having to be done later on. Now and again, in a private case, on the other hand, where treatment had been fairly well carried out for many years, the destruction of the ossicles had not been so great, but even there the result was not at all assured, and very often one had to perform the radical operation in the end, so that he said it was seldom wise to attempt to save the ossicles in middle ear suppuration. He had cases in which, when the ossicles were removed, the results with regard to hearing were quite as good as in the case operated upon by Dr. Logan Turner. The second question which had been raised had better be left for discussion by Mr. Ballance, who was present; but he might say he had practically given up grafting in the ear. If he had a large enough cavity, that was to say a cavity which did not become divided into, on the one hand, a middle ear cavity and on the other a false antrum, a bridge growing between, healing went on well enough without grafting. The kind of grafting indicated here was an old form of grafting; it was performed in the seventies by an American surgeon—at least it was found chronicled in one of the American books. These were the two points which seemed to him worth raising in this connection, and he would be glad to hear further discussion.

Mr. C. A. BALLANCE said that in the radical mastoid operation the ossicles should be left when they were not diseased. He knew this had long been the practice of Jansen and others. The bridge ought always to be removed, because otherwise disease might remain unremoved behind it or might recur behind it. It seemed to him that to remove the bridge and make all the cavities into one was the only proper surgical treatment for chronic disease. The other point raised by the last speaker was with regard to grafting. He might say that he always grafted. He had done many hundreds of operations and had always grafted the raw bone surface.

Dr. MILLIGAN thought that one of the best arguments for the removal of the bridge was the excellent result obtained where Nature had removed it in the course of the disease. He now almost invariably removed the bridge, but as far as possible preserved the ossicular chain. He gathered from Dr. Barr's remarks that what he specially wished to bring before the meeting was the method in which he applied the grafts in his various cases—

a method which he thought was not only novel but distinctly ingenious. With regard to grafting large cavities after the complete post-aural operation, his practice was to employ grafts according to Ballance's method. Not only does grafting materially shorten the process of after-treatment, but, so far as his experience went, he could not say that it had in any way caused a deterioration of hearing.

Dr. THOMAS BARR said in regard to Dr. Stoddart Barr's method of modified grafting that they had all experienced the difficulties associated with continuous packing in the after-treatment of the radical mastoid operation. While by this more limited method of grafting they did not get the complete effects of Ballance's method, there was no doubt that, in many cases, it very decidedly shortened the after-treatment. It was important to make good wide cavities, removing as far down as possible the posterior wall of the osseous meatus. He saw one of his son's cases a few days ago, three weeks and a half after the radical mastoid operation and two weeks after the partial grafting. The cavities were nicely lined with dry, pale pink epithelium, with the exception of the outer part of the post-meatal cavity, to which none of the graft had been applied; here there was some granulation tissue. It was interesting to see how the graft applied itself to the surface under the impact of the blowing of air through the glass tube. He could not say he had found simple syringing of the ear twice daily with the instillation of spirit very satisfactory in many cases without packing. His experience was that, owing to the formation of exuberant granulation tissue in the cavities, especially at the aditus, with coalescence, adhesions and pus-yielding recesses, one had to resort afterwards to curetting, cauterization and a measure of packing. Some of them, however, no doubt did well without packing.

DR. LOGAN TURNER said he had nothing to add but to congratulate Dr. Love on the results of the hearing after the complete mastoid operation if they were as good as had been obtained in the patient shown in the next room. He must also refer to the fact that the discharge in her case had lasted for twenty years, so that she had a very chronic condition.

Dr. PORTER, in reply, said that with regard to the skin grafting he thought there was a distinct alternative to this method of treatment and to packing. He referred to treating the cavity without any packing after the first week. Dr. McBride and himself had treated the mastoid cavity for eighteen months now after this method, and on the whole had been satisfied with the results. Of course one did not get success in every case, but no one could claim uniform success by any one method. In the earlier stages of healing the cavity certainly contracted very much, but some six months later the cavity, which at first seemed very narrow, had usually expanded, adhesions began to disappear, and the cavity assumed the typical kidney shape.

Dr. STODDART BARR, in reply, said he first of all wished to make it clear that any claim to originality that he made was confined to a few details in connection with the technique of the grafting operation. He was quite aware that, years ago, attempts were made to introduce thin grafts through the meatus, and that since then, from time to time, further attempts had been made, without, however, much success. The plan adopted by many surgeons to-day of treating the cavity made at the mastoid operation by syringing, followed by spirit instillations, had been far from uniformly successful in his hands. In many cases the cavity became filled up by granulations or subdivided into pouches or recesses by the formation of bridges or bands of connective tissue. It was in order to avoid these unfortunate results that he now, almost as a routine, carried out the grafting operation just described, and he had found that, even if the graft adhered only over the inner wall of the aditus and over the bridge, the spirit treatment could then be followed without any risk of the cavities becoming unduly obliterated.

A Patient after Vestibulotomy (Right Ear).

By J. S. Fraser, M.B.

W. B., male, aged 11, double otorrhœa after measles eight years ago. Six months ago tonsils and adenoids were removed, and two days later patient developed scarlet fever.

Examination on Admission.—Right side: Large aural polypus and profuse purulent discharge; mastoid tenderness. Left side: Granulations from posterior wall of bony meatus; no mastoid tenderness. Weber to right; Rinné negative, both ears; Schwabach lengthened. Watch, left $\frac{1}{60}$; right contact. Whisper, left: 18 in.; conversation, voice at 9 ft. Right ear: raised voice at 2 in. Patient cannot hear C2048 at all by right ear.

Static Organ.—Boy can stand with feet together and eyes shut, but not on either foot alone when eyes are shut; worse on right than on left side; on attempting to walk in a straight line with eyes closed he deviates markedly to right. Slight nystagmus on movement of eyes to left; marked nystagmus on syringing right ear with hot and cold lotion.

Operation (April 22, 1908).—Right antrum large, contained granulations which protruded through posterior wall of bony meatus; small amount of yellow, curdy pus in antrum. Bacteriology: bacillus belonging to *coli* group, producing gas and acid in glucose and lactose media.

Radical Mastoid Operation Performed.—Malleus removed; incus not seen; the external semicircular canal prominence was rough, and red in colour; it was opened, and the canal followed towards the ampulla, where an opening was made into the vestibule; no pus seen. The region of the oval window was found to be covered with small red granulations, and the stapes was absent; the vestibule was opened in this region by enlarging the oval window downwards and forwards; only clear fluid escaped in small quantity. Plastic on Körner's method; posterior wound closed with clips; granulations in left ear curetted. Duration one hour and a half.

Progress.—Vomiting continued for two days after operation; on fourth day wound cavity was dressed, but bleeding obscured the view; incision healed by first intention, but the boy was very noisy and restless during dressings, so that packing was rendered difficult; a fortnight after the operation packing was entirely discarded. One month after operation there is still slight nystagmus on rotation of eyes to left, and staggering when he stands on left leg with eyes shut, but both conditions less marked than before operation; patient giddy and inclined to vomit for one day after these tests had been applied.

Notes of a Case of Ablation of both Vestibules for the Relief of Vertigo.

By George Gibson, M.D., and Richard Lake, F.R.C.S.

The case, the notes of which we have the honour to bring before the Section, was originally shown before the Otological Society of Great Britain on March 6, 1905. The patient, a female, was then aged 26. She had had scarlet fever, rheumatic fever, and measles as a child. Her deafness commenced at the age of 19, though she had previous ear trouble, vertigo first being noticed about this time. When examined she was completely deaf to the voice, but was able to hear C^1 and C^2 when struck very hard.

On January 2, 1905, the whole of the inner ear on the right side was ablated. Some few months after this operation vertigo on the left side became so severe that the left ear was operated upon on January 17, 1906, and the vestibule ablated. There is nothing of note to report about the immediate after-result of this operation, and although at first one had been inclined to believe that the tinnitus had not been relieved by the first operation, this was not so, as the only noises the patient now hears are that occasionally she imagines that she hears voices calling her at night. Before the operation on the second side was undertaken, the advice of Dr. Risien Russell was sought, in order to obtain a definite opinion as to the probable effects of destroying the vestibular nerve on both sides. The form of operation adopted was fully described in the *Lancet* of January 6, 1906, so there is no need to trouble the members of the Section with any references to its technique.

Notes by Dr. G. Gibson's house physician, February, 1906:—

Nervous System. Subjective Phenomena.—The patient now complains of noises in the head only, which are only occasional, and also of some frontal headache coming on at irregular periods. This is sometimes relieved by epistaxis. She only occasionally has severe attacks of pain in the back of the head, but these have not been so bad since the last operation. Deafness is the only other subjective symptom.

Objective Phenomena. Cranial Nerves.—I. Sense of smell very good. Can distinguish pleasant from bad odours quite easily. No difficulty since operation so far as patient can tell.

II. Sight has never been very good, but has not got any worse. Both discs appear normal. Some myopic astigmatism, roughly about 2 D. or 3 D., in direction 15° to left. Some black specks are seen on lens on ophthalmoscopic examination, when the pupil is dilated close to the margin of the pupil. Nothing is seen by oblique illumination. Both fields of vision are normal.

III., IV., VI. All ocular movements are carried out at once, both eyes working well. There is no apparent weakness of any of the ocular muscles; no squint or diplopia. Well-marked nystagmus of a slow character is present.

V. There is nothing noteworthy. Sensation over face, as far as can be made out, is quite normal; no vasomotor or nutrition change.

VII. Right-sided facial paralysis. The whole right side of the face has lost expression, especially the forehead, where the wrinkles are lost on the affected side. The right palpebral fissure is smaller than the left. Mouth slightly drawn to the left. When the patient smiles the face is drawn to the left side. In frowning only the sound side is used. Blowing out the cheeks produces an escape of air on the affected side, but there appears to be a slight escape from the nose as well, indicating weakness of the palate. The right eye can be closed altogether, but not with the same force as the left. In fact all movements of the right face are greatly diminished.

Taste.—Patient to-night could not taste any of the solutions with either the sound or affected side. At times, while watching the right lower eyelid especially, a well-marked tremor has appeared, and one or two definite contractions of the orbicularis.

VIII. Complete deafness on both sides. The patient cannot hear a watch applied to the ear on either side, nor can she appreciate it if pressed on any part of her head. She can just make out a loud clap of the hands close to the ear, though not infallibly. All appertaining to coördination and equilibrium good. The patient walks wonderfully well, perfectly straight, without staggering and faltering, and can stand with her feet together both with eyes open and shut.

IX., X. No weakness of these so far as can be ascertained.

XI. Also seems quite sound. Both shoulders can be shrugged quite well, and patient says she has always been able to carry out this movement.

XII. When the tongue is protruded it is thrust out towards the left side, and there is considerable difficulty in getting it over to the right side at all. When examined in the mouth there is a scar on the right side, about anterior two-thirds, which diminishes the total size of this side. The patient cannot remember anything happening to cause this. The right side is much more tremulous than the left.

Common sensation appears to be very acute over all the limbs and trunk; nothing abnormal in sensibility to touch, heat and cold, and pain; some slight discharge from left ear. Muscular sense normal. Pupils equal, quite round and regular, and react to light and accommodation; all organic reflexes are carried out normally; no difficulty in swallowing or breathing. Plantar very lively indeed, but of ordinary flexor type; no Babinski; abdominal reflexes also normal; ankle-jerks present; knee-jerks, with markedly increased clonus, obtained in right one; no definite ankle-clonus; supinator-, triceps-, and biceps-jerks all markedly present on both arms, somewhat exaggerated on left side, and at one time the biceps went into clonus for a short time. Voluntary movements can all be carried out quite well except as mentioned above in face. Coördination, gait, &c., perfect. Electrical reactions of face give no reaction of degeneration. No evidence of any vasomotor change over any part of the body, such as face. Intelligence, attention, both very good; some little difficulty in remembering details at times. Speech somewhat indistinct, patient slurs words at times. Is quick at picking up spoken words from the movement of the lips, and these are answered well.

Note by Dr. George Gibson: Professor Schäfer was kind enough to associate himself with me in making some observations in regard to the patient's powers of equilibration, as well as on the strength of her muscles. When placed upon a revolving chair, by means of which she could rotate accurately round her own vertical axis, we found that she was absolutely unable to detect even very considerable degrees of rotation. Unless the chair was turned round so quickly as to give a distinct sense of resistance to the skin she was not aware that she was being turned round at all. We tested this point upon ourselves in order to have some comparison and found that the very slightest rotation was detected by each of us. We had the advantage, when making these observations, of the supervision of Professor Crum Brown, whose researches upon the semicircular canals are so widely known. Professor Schäfer further tested the strength of the patient's upper extremities by means of the ergograph. The most careful observations failed to detect any departure from a normal standard, and there was absolutely no difference between the two sides. It may be of interest to add that a youthful patient, who was lately in Ward 30 of the Royal Infirmary under my care, suffering, amongst other affections, from congenital deafness due to inherited specific infection, provided an interesting subject for control observations. Through the kindness of Professor Schäfer, Mr. Ednie, of the Physiological Department, assisted me in testing this patient with the rotating chair. It was found that, even when turned round with considerable velocity, she was quite unable to appreciate the fact of rotation.

Note by Mr. Richard Lake: I will only deal briefly with my experience of this operation for the relief of vertigo. I brought forward the first recorded case in March, 1906, before the Otological Society of Great Britain. Since then I have operated for vertigo in Ménière's disease, unconnected with suppuration, five times—six in all. These cases were all operated on for the relief of a condition which either caused agoraphobia or prevented the patient from earning his or her livelihood. In all complete relief has been afforded—in the first case for four years, in the second, third, and fourth for periods of one to three years, in the last two one under six months and the other only for as many weeks. The one which was only done four months ago has, I understand, undergone another operation for the relief of noises, which, as they did not appear to me to be of aural origin, I much preferred not to attempt to relieve.

DISCUSSION.

Dr. MILLIGAN said, with regard to Dr. Fraser's case, the very important point was raised as to how far one was justified in opening the labyrinth where there was simply a bony erosion of the external semicircular canal. After reading the notes of the case it appeared to him that there was not sufficient reason for the labyrinth to have been opened. The lesion appeared to have been superficial and limited to the bony capsule of the canal, and he thought that under such circumstances all that was necessary was to perform the complete post-aural operation and to watch the subsequent development of events. As labyrinthine surgery for suppurative disease was as yet in its infancy it seemed to him most important that one should very carefully weigh the indications for opening the internal ear and that no routine method should be adopted. With regard to the case recorded by Dr. Gibson and Mr. Lake he would like to offer his congratulations upon the success of their endeavours. The result he considered a great triumph in surgery.

Dr. ALBERT GRAY said he would like to say a few words about the physiology of the vestibule and canals and the light thrown upon it by a case such as this. Ewald, some years ago, put forward a theory to the effect that these structures were responsible for the tone of the skeletal muscles, and he described an extraordinary number of experiments to support that view. He noticed, from the physiological examination of this patient after operation, that the case did not lend any support to Ewald's theory at all. The case rather corroborated the view that these structures were concerned in giving information as to the position of the head and, to a certain extent, the position of the body after rotation. That was Crum Brown's theory, expressed a great many years ago, and he believed that it was the one which was most satisfactory. His own investigations of the comparative anatomy of the subject also seemed to give evidence in support of the correctness of Crum Brown's theory, for in animals that have their cervical vertebræ ankylosed, such as the Cetacea, the movements of the head were extremely limited; and associated with this was the fact that in these animals the semicircular canals and vestibule had undergone extraordinary retrograde changes and the nerve supply was very much reduced.

Mr. SYDNEY SCOTT said that Dr. Fraser's case was one which interested him very much, especially as he noticed the operation on the labyrinth was that described by Dr. Milligan as the "bridge operation on the labyrinth" and which, with Mr. West, he described under the name of "double vestibulotomy," as distinct from other forms of vestibulotomy, in which a single opening is made in the vestibule. He welcomed the case which Dr. Fraser had frankly put forward as a contribution to our study of the surgery of the labyrinth. They wanted to know all they could about these operations, their precise clinical indications, as well as the lesions with which they were associated. In particular they would like to know something more about nystagmus and the connection between nystagmus and intralabyrinthine lesions. Bárány's observations were, he believed, well known. Although unacquainted with the methods of the Viennese school it so happened that he had taken the opportunity of examining a considerable number of cases for nystagmus, and it had now become a matter of routine to observe the exact forms of nystagmus met with in aural diseases. He would suggest that in descriptions of cases in which the term "nystagmus" was used they should pay special attention to the form and direction of the movements constituting nystagmus. He would like to ask Dr. Fraser what was the direction of the rapid movement of the eyeballs in the nystagmus provoked by syringing the right ear of his patient before the operation with hot water, and whether the movement differed in direction after syringing with cold water. He had been able to bear out the principal observations of Bárány with regard to the effects of syringing with hot and cold water, supposing the outer wall of, for example, the *right* labyrinth was exposed by a defect in the tympanic membrane to the influence of heat and cold: when the labyrinth was normal a definite form of nystagmus could be produced. On syringing the ear with head erect, taking care not to exert increased pressure on the oval window, using water at 110° F., or, if there was no response, gradually increasing the temperature of the water to 112° F. or 115° F., or in some cases even to 118° F. (which was the limit of toleration), a certain form of nystagmus resulted. When the gaze was directed to the right, that is the syringed side, the rapid rhythmic movement of the eyeballs was directed horizontally to the same side; this was often accompanied by a slight rotatory movement downwards. On fixation of the eyeballs to the extreme left the nystagmus absolutely disappeared, to return when the gaze was once more turned to the syringed side. There were some cases in which the nystagmus was very violent and was not arrested even in extreme deviation of the eyes to the opposite side, but the direction of the *rapid* movement was then always toward the side affected by heat. Cold water produced a different result. On syringing with water at 90° F., sometimes only after decreasing the temperature gradually to 80° F. or 70° F., or even 65° F., he had found that after an appreciable interval of time nystagmus, if present as the result of hot syringing and manifest during deviation of the eyes towards the syringed side, first ceased and then became replaced by nystagmus which was manifest on deviation of the eyes towards the side opposite that which was syringed. The rapid movement of the eyeballs was towards the opposite side and was either horizontal or rotatory when the head was erect; that was to say, the nystagmus produced by cold was of the same type as that produced by heat, but the direction of deviation in which the nystagmus became manifest, as well as the direction of the *rapid* movement, was towards the opposite side. He had purposely laid stress on this question of nystagmus because it had been said to be rarely of much value, whereas his own observations led him to place very high value upon its significance. In gross lesions or destruction of the labyrinth he had failed to elicit nystagmus by either heat or cold. His plea was for more precise information on this important subject of nystagmus.

Mr. **HUNTER TOD** said there was one practical point he would like to ask about, namely, what was really meant by "ablation" of the vestibule? Did some include in this category the simple opening of the vestibule by chiselling away a small fragment of the external semicircular canal; or was it restricted to "ablation" in its true sense, meaning complete removal of the semicircular canals and curettement of their openings into the vestibule? To determine the value of operations on the labyrinth, and whether they should be considered dangerous or not, it was, in his opinion, very important that in the description given of any such operation the actual extent of the operation should be clearly defined, otherwise a false impression might unintentionally be given as to what was really done.

Dr. **SYME** said there seemed at the present time a danger of the hasty opening up of the internal ear when symptoms pointing to labyrinthine involvement arose in the course of middle ear suppuration. This operative procedure was attended with a good deal of risk. Moreover, in the great majority of cases the condition of the labyrinth was one of congestion or of inflammation and not of actual septic infection, and the symptoms disappeared after the performance of the radical mastoid operation. It was not unusual to find erosion of bone in the region of the external canal or of the inner tympanic wall, but with this was also frequently a protective formation of bone shutting off the cavities of the internal ear; so that their practice should be to perform the radical mastoid operation and, unless some very decided indication for opening up the internal ear, such as the escape of pus from a sinus, was found, to wait and watch the case carefully. If it was decided to proceed further the whole labyrinth should be laid open, but this, he thought, would be very rarely called for.

Mr. **A. L. WHITEHEAD** thought that in many cases of mastoid disease with a fistulous opening into the external semicircular canal, uneventful and perfectly satisfactory healing occurred after the performance of the radical mastoid operation alone. In Dr. Fraser's case it might have been desirable to try first the simple mastoid operation before opening up the entire labyrinth.

Dr. **KERR LOVE** said that when one heard that in twenty or thirty hospital cases operation on the internal ear had been performed, and when one could not find in ordinary hospital practice cases corresponding to these, one suspected that the internal ear operation was being unnecessarily performed. On the other hand, cases occurred in which special interference with the canals was undoubtedly an advantage, but as a rule it was not necessary to obliterate the whole internal ear.

Dr. McBride wished to add his congratulations to Mr. Lake on this case and the others to which he had briefly referred. He hoped that on another occasion he would give fuller details. With regard to Dr. Fraser's case he had also intended to ask him why he operated on the labyrinth, but as this had already been done he had nothing further to add.

Dr. Fraser, in reply, said he had been pretty generally attacked for having performed this operation. He could only say that he did so on the symptoms. The patient was decidedly giddy and, with his eyes shut, could not stand upon either leg alone nor walk straight. Of course none of them were called on to do this, but still there was no doubt that, in this case, there was interference with the balancing powers. He quite admitted that it was possible that this patient would have got well if the radical mastoid operation had been performed and nothing further done: but he maintained that he had been justified by the result, as the patient was now much better. It was before his time, but he thought he was safe in saying that the arguments they had just heard were probably used when the radical mastoid operation was first introduced—he meant to say that at that time conservative operators regarded it as unnecessary to go clearing away the "bridge," the outer wall of the attic, the malleus and incus when they had got very nice results with the Schwarze operation.

Notes of a Case of Otitic Meningitis, with Histological Specimens (Lantern Slides) of the Labyrinth, demonstrating Streptococci in situ.

By Sydney Scott, M.S.

Notes.—Primary streptococcal tonsillitis and pharyngitis, followed by acute otitis media, labyrinthitis, and lepto-meningitis.

Boy, aged 3½, admitted to the Evelina Hospital for Sick Children under the care of Dr. Whipham, March 31, 1908.

History.—There had been diarrhœa and vomiting lasting one day a week previously, and difficulty in swallowing had been present for four days. No rash.

Condition on Admission.—Child looked very ill. Apathetic appearance. Temperature 102° F., pulse 140, respirations 36. Fauces and uvula ulcerated and covered with slough. Bacteriological examination by Dr. Leathem, pathologist to the hospital, showed predominance of long-chained Gram + (positive) cocci on swabs from throat.

Two Days after Admission.—The child was seized with general convulsions, followed by loss of consciousness. He remained more or less deeply unconscious from this time onwards. There was no paralysis.

Nine Days after Admission.—General condition unaltered. Slight purulent discharge was first noticed from the left ear.

Seventeen Days after Admission.—I was asked to see patient on account of the otorrhœa. I found the right ear normal. The fundus of the left external auditory meatus contained pus and swollen mucosa, which obscured the defect in the membrane. No superficial mastoid swelling. No facial paralysis. Very slight head retraction. Lumbar puncture showed excess of cerebrospinal fluid, but no abnormal cells or organisms could be found after centrifugalization. Leucocyte count 7,000. The optic discs were normal. Slow, continuous wandering, conjugate movements of the eyeballs could be seen, but there was no spontaneous nystagmus. Knee-jerks equal. No ankle-clonus. Plantar reflex—flexor response. Kernig's sign absent.

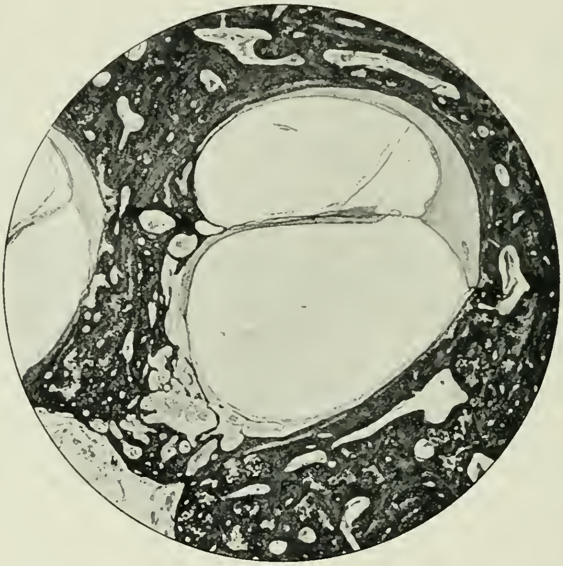

Fig. 1.

Transverse section of lowest coil of normal cochlea from child at birth. Magnification 20 diameters.

Operation.—I performed the radical operation the same day. The cavities of the middle ear and antrum contained inflamed mucosa, no pus. There was no extra-dural abscess. The outer wall of the labyrinth was normal in appearance; there was no erosion of the external semicircular canal or promontory. The stapes was intact in the fenestra ovalis. The mucosa lining, the fossula rotunda, and sinus tympani was greatly swollen. The posterior cranial fossa and the intra-dural space in front of the cerebellum were explored with a director, and much cerebrospinal fluid escaped and continued to drain away. Bacteriological examination of this fluid, collected during life, revealed Gram positive streptococci.

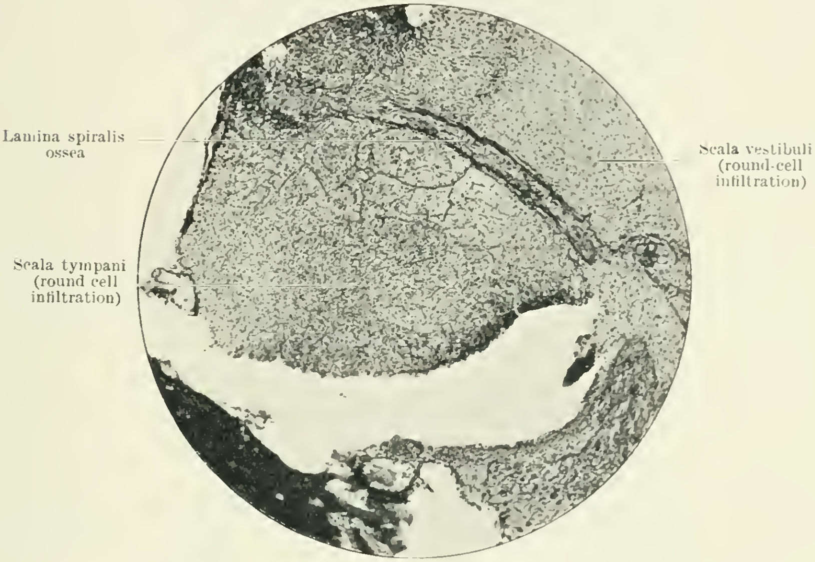
Lamina spiralis ossea
Scala vestibuli (round-cell infiltration)
Scala tympani (round cell infiltration)

Fig. 2.

Section of cochlea from a case of diffuse streptococcal labyrinthitis. Details of the case from which this specimen was obtained have been published in the *Archives of Otology*, 1908, vol. xxxvii., No. 2, "A Case of Acute Internal Hydrocephalus Secondary to Streptococcal Infection of the Labyrinth." Magnification 60 diameters.

Result.—After the operation no improvement followed. The temperature rose to 108° F. Several general convulsive seizures preceded coma, and death followed two days afterwards.

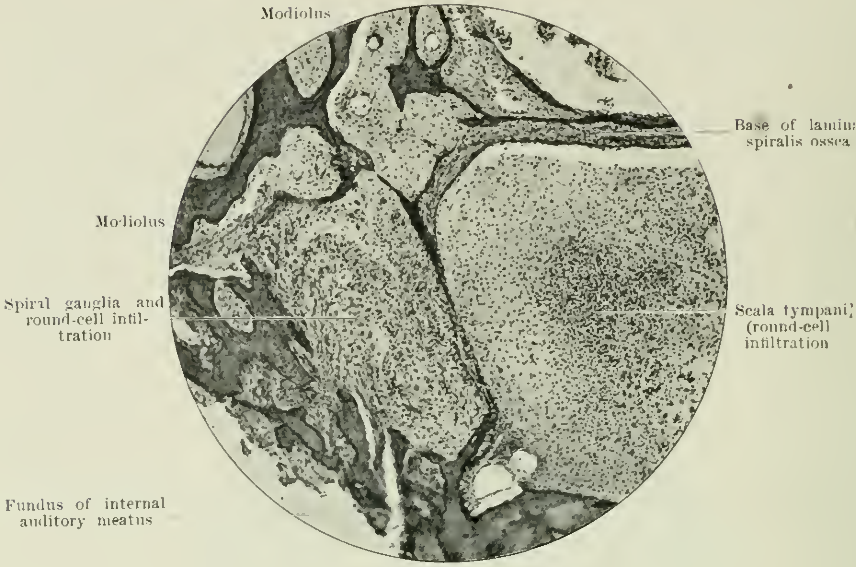
Modiolus
Base of lamina spiralis ossea
Modiolus
Spiral ganglia and round-cell intiltration
Scala tympani (round-cell infiltration
Fundus of internal auditory meatus

FIG. 3.

Similar section of the cochlea from an unreported case of lepto-meningitis secondary to chronic suppurative middle ear disease. Magnification 60 diameters.

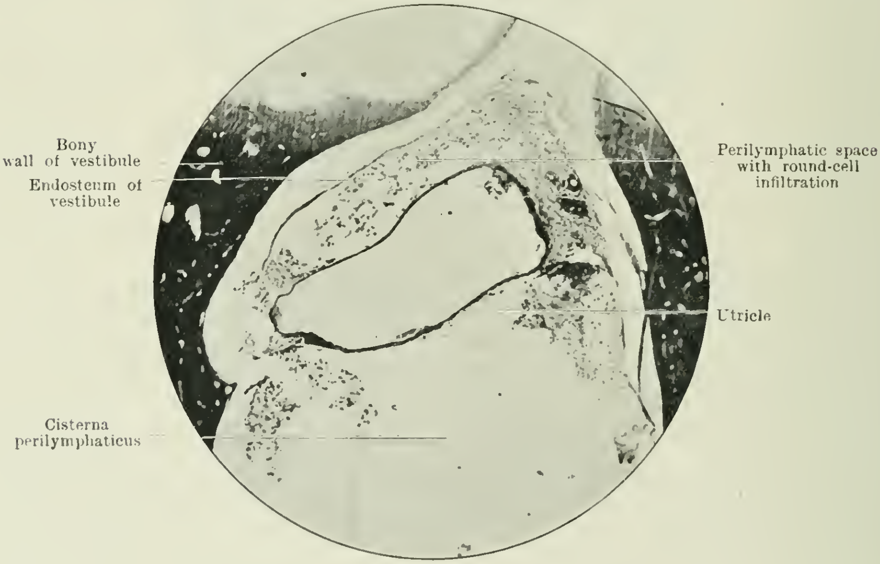

FIG. 4.

Section through vestibule and utricle from the same case as that supplying the previous specimen. Magnification 20 diameters.

The Post-mortem Examination.—Excess of turbid cerebrospinal fluid was found within the lateral ventricles of the brain, and a little flaky exudate around the medulla and pons. In this fluid were found Gram positive streptococci and very few polymorphonuclear cells. To the naked eye there was no other intracranial lesion; the right temporal bone appeared perfectly normal, and nothing abnormal was found bearing on the subject elsewhere in the body. I examined the *left* petrous bone histologically, and found the labyrinth to be in a state of acute diffuse round-cell infiltration, and containing swarms of long-chained streptococci staining by Gram's method (fig. 6).

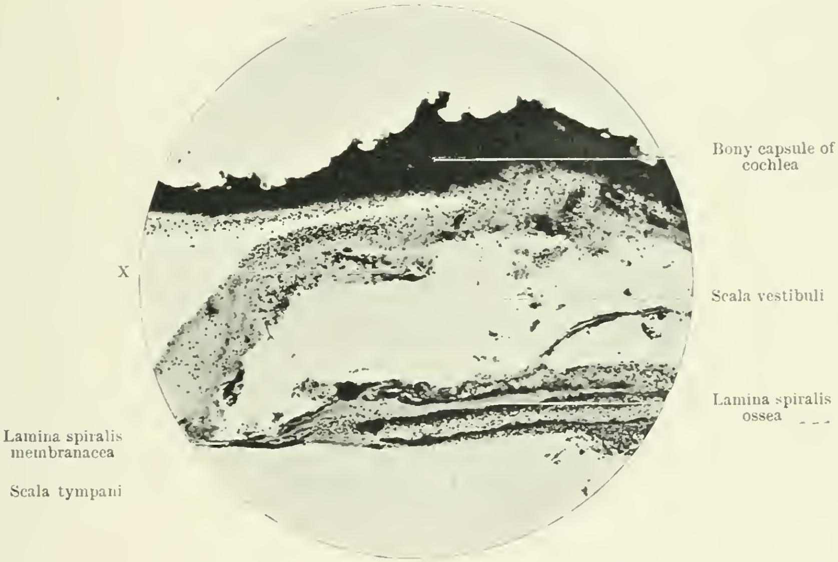

FIG. 5.

Section of the cochlea from the case the notes of which were before the members of the Section at this meeting. Magnification 60 diameters.

X Area of section shown in fig. 6.

I venture to submit that cases of this description are not rare that the meningitis, especially in children, is due to unsuspected unilateral labyrinthitis, secondary to middle ear disease. A consideration of such cases which have come under my observation leads me to infer that many should be saved by timely operation, that the distended cisternæ at the base of the brain should be drained *through the labyrinth*, and the spinal theca drained by lumbar puncture; possibly it would be necessary in some cases to drain the lateral ventricles too.

Note.—I am indebted to Dr. Whipham for permitting me to record this case.

The accompanying lantern slides of microphotographs of histological preparations of the human labyrinth show the changes produced by acute inflammation. (Microphotography by Dr. Albert Norman, of London.)

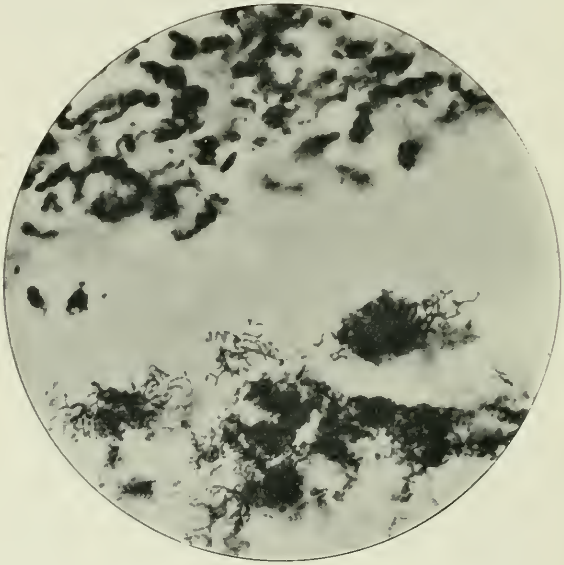

FIG. 6.

View of the same specimen magnified 800 diameters and stained by Gram's method, showing crowds of leucocytes and masses of long chain streptococci in the scala tympani of the cochlea. Thickness of section = 3 micromillimetres. (Cut in paraffin.)

Preliminary Note on Cytological Examination of the Discharge in Cases of Middle Ear and Maxillary Sinus Suppuration.

By JOHN M. DARLING, M.B.

IN ten cases of suppurative middle ear disease a cytological examination of the pus was made. Leishman's and Jenner's stains were used. The results coincided for the most part with those of Dr. Milligan. The presence, however, in some instances of large numbers of epithelial squames suggested cholesteatoma where at subsequent operation none was found.

In forty cases of maxillary sinus suppuration a similar examination was made. The pus was usually obtained by puncture of the nasal wall and washing out. Recognition of the different types of cell was rendered difficult as a rule by the advanced degeneration present. More satisfactory results would probably be obtained by examination of a second washing at a short interval after the first.

In thirty-one chronic cases no myelocytes were found. Five showed lymphocytes in excess, and epithelium was recognized in sixteen (one ciliated, four columnar, nine squamous, and two both columnar and squamous).

Nine recent cases showed polymorphonuclear and mononuclear pus cells only, with the exception of one case—a nine months case already under treatment for three months by the nasal route—where squamous epithelium was observed in addition.

DISCUSSION.

Dr. LOGAN TURNER said he would just like to say that he had been watching Dr. Darling's work in connection with the cytology of maxillary sinus discharge in the hope that they might learn something to help in the treatment of these cases. Unfortunately, he thought he was right in saying that the results obtained by cytological examination were not very encouraging so far, but Dr. Darling would himself state the result of his investigations.

Dr. DARLING, in reply, said his object had been to carry out, in connection with the cytology of maxillary antrum discharges, an investigation on lines parallel to those followed by Dr. Milligan and Dr. Wyatt Wingrave in connection with ear discharges. The difficulties had been mainly that it was extremely difficult to get a specimen of pus from the antrum uncontaminated, and that it was very often extremely difficult to make out the various kinds of cells in the pus, because the degeneration was so very marked. Of the thirty-one chronic cases he had examined, only sixteen had shown epithelium and five leucocytes in excess, while none had shown myelocytes. The one case in which ciliated epithelium was found was cured by operation through the nasal wall. Of the four cases which showed columnar epithelium one had not yet been treated, two were treated by the alveolar operation and were still uncured, and one was cured by radical operation. The two cases where both columnar and squamous epithelium were found were both cured by operation by the alveolar route. Of the nine cases showing squamous epithelium only, five were treated unsuccessfully by the alveolar or nasal operation, two were cured by the alveolar operation, and two by the radical operation. One of the cases which showed lymphocytes in excess was cured by radical operation, while the other four treated by the nasal route were still uncured. Of the nine recent cases which he had examined only one showed epithelium. Two were cured by radical operation and the remainder by measures short of that, with the exception of the case where epithelium of the stratified type was found. This case was treated by the nasal route and was still uncured. The results encouraged one to proceed further, but he did not think that they had as yet any definite data to act upon.

Dr. ALBERT A. GRAY showed: *Stereoscopic photographs illustrating the comparative anatomy of the labyrinth of reptiles, birds and mammals.*

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE FIRST

COMPRISING THE REPORT OF THE PROCEEDINGS FOR THE
SESSION 1907-8

PATHOLOGICAL SECTION

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PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE

PATHOLOGICAL SECTION.

CONTENTS.

October 15, 1907.

PAGE

Presidential Address. By S. G. SHATTOCK 1

An Example of Incomplete Glandular Hermaphroditism in the Domestic Fowl. By S. G. SHATTOCK and C. G. SELIGMANN 3

A Case of Macroglossia neurofibromatosa. By W. G. SPENCER and S. G. SHATTOCK 8

November 19, 1907.

A Contribution to the Study of the Relationship between Avian and Human Tuberculosis. By S. G. SHATTOCK, C. G. SELIGMANN, L. S. DUDGEON, and P. N. PANTON 15

Some Experiences with the Tuberculin Ophthalmic Reaction. By L. J. AUSTIN and OTTO GRÜNBAUM 74

Abscess of Bone caused by an Intermediate Bacillus (?) allied to *B. paratyphosus*. By F. G. BUSHNELL, M.D. 79

January 21, 1908.

A Remarkable Case of Diffuse Cancellous Osteoma of the Femur, following a Fracture, in which similar growths afterwards developed in connection with other bones. By W. H. BATTLE and S. G. SHATTOCK 83

Tuberculous Endocarditis. By W. O. MEEK 116

PAGE
Sarcoma of the Rectum in a Boy aged 10. By CECIL ROWNTREE 124
On the Action of Vesical Calculi upon a Photographic Plate in the Dark. By HECTOR A. COLWELL 127

February 18, 1908.

Acidosis in Pregnancy. By J. B. LEATHES 131

March 17, 1908.

Three Specimens of the Larvæ of *Eristalis tenax*, passed by the Bowel. By S. G. SHATTOCK 147
The Influence of the Testis upon the Secondary Sexual Characters of Fowls. By C. E. WALKER 153
A Case of Congenital Defect of the Muscular System (Dystrophia muscularis congenita) and its Association with Congenital Talipes equino-varus. By RUSSELL HOWARD 157

The Relations of Endothelioma to other Forms of New Growth:—

(1) General Survey. By W. S. LAZARUS-BARLOW 167
(2) The Occurrence of Lumina in Malignant Tumours. By L. COURTAULD and A. LEITCH 173
(3) The Significance of Prickle Cells in the Identification of New Growths. By CECIL ROWNTREE 175

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Pathological Section.

October 15, 1907.

Mr. S. G. Shattock, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

In the evolution of Pathological Science it is an incontestable historical truth that its starting-point is morbid anatomy. The systematic study of the changes discoverable by the naked eye has preceded every other form of observation, for reasons as obvious as they are natural. The great work of Morgagni (1682-1771), "De Sedibus et Causis Morborum per Anatomen Indagatis," is purely anatomical; it contains no reference to any microscopical investigation. With the termination of Virchow's labours in the succeeding century (1821-1902) we had almost come to think of a completion or consummation of morbid histology, the method of investigation which stands next in evolutionary order. Not that either of these fields, of course, is exhausted, but the area of each is so reduced as to make further discoveries increasingly difficult.

Ord's recognition, nevertheless, of the association of thyroid atrophy with the clinical condition of myxœdema is within the memory of all; yet it is as simple a piece of morbid anatomy as can be well imagined, not less simple, indeed, than the observation of Addison in regard to the adrenals. And both discoveries have given rise to experimental and histological investigations of the highest importance.

The histological discovery of deciduoma malignum and of chorionic epithelioma of the testicle will further prove that in morbid histology (apart from cytology) there are still new observations to be made; not to speak of the additions to our knowledge of the morbid histology of the blood, following largely upon Ehrlich's methods of investigation. It is a favourite belief of mine that there will always be a new disease for any one to discover who seriously undertakes the task of differentiating it from the number of undiagnosable and obscure conditions, both clinical and pathological, which so frequently obtrude themselves into notice.

We have, however, long ceased to regard morbid anatomy and morbid histology as constituting the whole of Pathology. In the great majority of cases they form *ipso facto* the object-matter of clinical diagnosis, and their importance from this standpoint will remain unassailable. But our present conception of Pathology is that of a science which must be studied from every side. Its additions have not infrequently been made from wholly unexpected quarters. The unity, by which we mean the unbroken continuity, of Nature absolutely forbids us to put a boundary between any single science and any one of the rest.

Specialisation is, it is true, imperative. But we must not, on that account, allow ourselves to regard it as a thing to be praised and admired. It is, on the contrary, a humiliating compromise, in spite of, or rather because of, its necessity. And the reason is plain. It accentuates divisions amongst phenomena where there are no such divisions in Nature. We cannot recognise a Pathology from which clinical observation and physiology, any more than botany, and zoology, and physics and chemistry, are excluded. It would be something altogether monstrous, like the most defective forms of a concrete teratology, and equally horrid and unviable.

But in this metropolis, possessed as it is of so vast an amount of clinical material, it is especially fitting to investigate disease in its more immediate relation to Medicine. Together Pathology and Medicine may be studied with the highest mutual advantage; to divorce them would be disastrous to the progress of both. This is, it seems to me, the side that admits of particularly advantageous study; not, of course, to the exclusion of any others, but amongst them, for all are equally welcome.

And now we have, as members of the Pathological Section of the Royal Society of Medicine, to be loyal to its objects and its interests. And these will be chiefly forwarded by such as are engaged in research, or who have the opportunity of making observations, contributing to our Proceedings. It is upon what will appear in these that the position of this Section, and, in a proportionate degree, the position of the Society as a whole, will depend. It is in our own hands to build up or leave unbuilt. We cannot lay the responsibility elsewhere; it rests absolutely with ourselves.

And lastly, in regard to the discussions or remarks made upon communications. It has been hitherto the practice, after the communication of a paper, for subsequent speakers to make their observations in a set or continuous form. In place of this, I propose substituting, during the year of office with which you have so unduly honoured me, another method of discussion, which might by contrast be called that of interruption—the method which persons adopt in discussing any subject privately. Its advantages are, I submit, that it will encourage debate by making it so much easier to take part in it; it will elicit opinions more effectively, and it will be less tedious to those who listen.

I do not see why a third or even a fourth speaker might not join in and interpose remarks. The method, in short, is that which is commonly spoken of as the Socratic, as we know it in the immortal Dialogues of Plato. And surely no higher guarantee than this is needed of its value. It is the method particularly devised, as being the one best adapted, for sifting scientific questions.

An Example of Incomplete Glandular Hermaphroditism in the Domestic Fowl.

By S. G. SHATTOCK and C. G. SELIGMANN.

IN the fifty-seventh volume of the *Transactions of the Pathological Society* we have described at length a case of true hermaphroditism in the domestic fowl. In the present communication we venture to record a second example, although its anatomical grade is decidedly lower than that already referred to, since the sexual gland, whilst it presents tubuli of male and female type, does not exhibit any area in which spermatogenesis or ovulation is in progress. The bird is a Leghorn fowl, which was kindly given to us alive, in January, 1907, by Dr. Leonard Hill, who has furnished us with the following history:—

It was bought with eleven young white Leghorn hens three summers previously, and at that time attracted no attention for the first six months or so, the bird presenting no features differentiating it from the others. Other cocks were seen to put their wing out and run round it as if about to tread it; and it was observed to tread, or endeavour to tread, a hen three or four times. It was never heard to crow, or to make the attempt.

When we received it, it was turned out with a game cock and a white Leghorn capon which had been brought up together and lived on amicable terms. The cock dropped his wing and waltzed round the newcomer, but did not molest it. The next day, however, he suddenly attacked it and inflicted injuries to which it succumbed shortly afterwards.

The tail of the bird is quite feminine, and altogether devoid of sickle feathers. The comb is that of a hen or young cockerel. The neck hackles are somewhat longer than normal in the female, but not pointed as in the male. The legs are provided with spurs. These are about 1·8 cm. in length, somewhat slender, but very sharp.

On dissection, a well-developed oviduct was found on the left side; it was normally pervious at its upper and lower end. There was no oviduct on the right. No vas deferens was discoverable on either side. The sexual gland was single and lay to the left of the middle line. It measured about 1·3 cm. in the chief, vertical diameter, and presented an obviously abnormal configuration. The surface is throughout devoid of the vesiculation indicative of the presence of ova. The gland is quite solid and in different degrees lobulated. The chief lobule attains a maximum diameter of ·5 cm. The depth of the lobulation varies; the larger lobules are almost pedunculated; in the lower part of the gland the surface is hardly more than lowly mammillated.

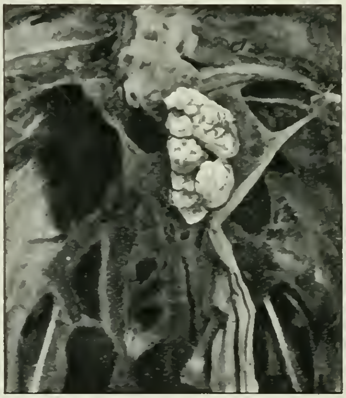

FIG. 1.

Showing the imperfectly developed sexual gland of the Leghorn fowl described. The gland presents itself as a solid, non-vesiculated structure, in different degrees lobulated, and occupying a position on the left side of the mid-line. In addition to the gland, there is shown the upper end of a well-developed oviduct. Natural size.

Histology.—Serial sections of the summit of the gland were made, and stained with hæmalum and eosin. As studied in the horizontal sections, the gland is connected dorsally with the front of the aorta, and loosely with the adrenal. It is composed of interconnected lobules, the structure of which is of three kinds. In one the glandular element is of the female type, in another of the male; while in the third no formed sexual elements are present.

(1) To take the last-mentioned first, in order to dispose of the least interesting. The substance of such lobules consists of a richly-celled connective tissue, so extremely vascular that it might almost be called

cavernous. In addition to the maze of vascular clefts, numerous arterioles lie in the tissue. No epithelial structures are discernible in the stroma.

(2) The lobules which represent the female constituent of the gland exhibit the following structure: Supported by a scanty basis of similar highly cellular connective tissue, there is a close plexus of epithelial elements. The latter elements are grouped together in solid columns, which are nowhere broad; some, indeed, are quite narrow, and comprise in cross section not more than three closely adpressed cells. The individual cells of the columns are devoid of any definite form, and the columns in no instance present any trace of lumen.

This cell plexus reaches the free surface of the lobule, though a thin layer of cellular connective tissue intervenes between it and the investing epithelium, which is represented by a single layer of cubical cells. From their general characters it would appear that these epithelial columns represent Pflüger's tubes. We have not, however, seen any ova developed in connection with them.

In some of the lobules the cells representing a female constituent have a less regular disposition than described; the cells are distinguishable from those of the stroma by their size and grouping, but the columns are less well defined and comprise fewer elements.

(3) In one lobule of particular size the epithelial tissue presents a different character. In a very scanty stroma there lie well-developed tubuli of notably larger size than the solid columns already described as Pflüger's tubes. The tubuli are closely arranged and tortuous, with scarcely any intervening stroma, as in a testicle. They present a lumen, and are lined with long columnar cells with basal nuclei. At the free surface of the lobule there is a thin zone of cellular connective tissue invested with cubical epithelium. The tubuli approach closely to the surface, but no continuity with the investing epithelium is discoverable.

Although the size of the tubuli varies, and the smallest do not present a lumen, nevertheless the individual cells preserve a size exceeding that of the elements which compose the columns in the female constituent.

The particular lobule described lies in the mid-line of the gland and projects at the free ventral surface from between its neighbours. The position of it, even apart from its structure, precludes the possibility of its being epididymis or parovarium. In addition to the chief lobule of this type, which projects from the ventral surface of the gland, there is on either side of it one more deeply placed, of lesser size, but presenting a similar histological structure.

At the extreme lateral border there is, contained in a circumscribed area of connective tissue, a small group of compactly arranged tubuli, which contrast with those already described, in greater regularity of size, in a more sharply defined lumen, and greater clearness of the columnar cells which line them. This body may be regarded as parovarium or epididymis indifferently. The large size of the blood spaces, the fact

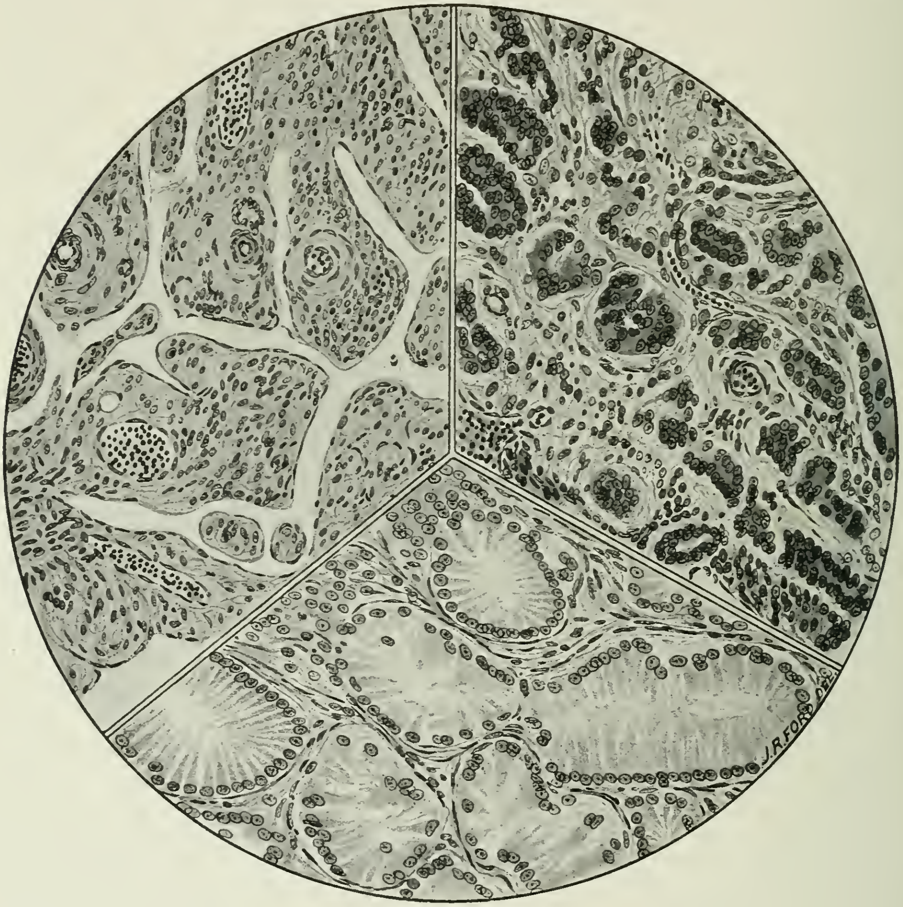

Fig. 2.

A combined figure, in the three sectors of which are shown portions of different lobes of the ill-developed sexual gland of the bird described in the text. The magnification throughout is the same, viz., one-sixth objective. In the upper left-hand sectors is shown portion of one of the lobes devoid of sexual structures. It consists of a highly vascular or cavernous richly-celled connective tissue, without any epithelial elements. In the upper right-hand sector of the figure is shown portion of a lobule, the stroma of which supports cell columns, suggestive of Pflüger's tubes. In the lower sector is portion of another lobule in which there are tubuli of conspicuous size, provided with a lumen, and lined with columnar cells, suggestive of testicular tissue.

that some of these lie not in the substance of the tubular tissue but at its surface, and the presence of a redundance of appertaining loose connective tissue, all point to the same conclusion. Although this bird may be classed as hermaphrodite from the structure of the sexual gland, there is no anatomical evidence that either spermatogenesis or ovulation ever took place.

In the hermaphrodite Leghorn already described by us in the fifty-seventh volume of the *Transactions of the Pathological Society* there were two sexual glands separated by a median fold of peritoneum. And we may most readily present the chief features of this interesting example by briefly quoting from the account already published. Of the two oviducts present, that of the left side is fully developed and has a normal disposition, its upper end being widely patent; the right is diminutive. The sexual gland of the left side is of flattened form, about 3 cm. in chief vertical diameter, of pale yellow colour, and presents a convoluted surface like that of a cerebrum in miniature. The sexual gland of the right side is considerably smaller, but presents similar microscopic characters. On each side there is a vas deferens. The larger gland of the left side has a typically tubular structure. The cells which line the tubuli are prolonged centrewards, so that, notwithstanding the fact that nearly every tubule is of conspicuous diameter, it has no proper lumen, but is filled with a coarsely vacuolated substance, produced in part at least by the inner portions of the cells. The stroma between the larger tubuli is scanty, but conspicuous groups of polyhedral interstitial cells occur in it. In the serial sections made from the lower end of this, the left, gland two ova were found.

In regard to the gland of the right side, it presents a tubular structure like the left; but, lying amidst the general collection of inactive tubuli in one of the convolutions of the gland, there is a microscopic area in which the tubuli are of conspicuously larger diameter than elsewhere, and distended with closely-packed epithelial cells, the nuclei of many of which present mitotic figures. In the more central cells of one of these active tubuli spermatogenesis is in progress.

Both of these birds appear to us as of interest in their bearings upon a theory we have advanced (*loc. cit.*)—namely, that the transformation of plumage in the female of certain birds to that of the male, accompanied as it sometimes is by the cessation of laying and the acquirement of male instincts, indicates an hermaphroditic condition of the sexual gland, the *male* constituent of which commences to functionate after the atrophy of the *female*.

A Case of Macroglossia Neurofibromatosa.

By W. G. Spencer and S. G. Shattock.

The first case of this remarkable condition recorded is that read on October 21, 1902, and published in the *Transactions of the Pathological Society*, vol. liv., 1903, by one of us (S. G. Shattock) in conjunction with Mr. F. C. Abbott. That which it is proposed to describe in the present communication is in its main features remarkably like the first, except that the patient is an adult, and that the diseased organ is considerably larger in an absolute, if not a relative degree.

Very briefly to re-state the first case, it concerned a child, four years of age, who was admitted into St. Thomas's Hospital under the care of Mr. F. C. Abbott. Swelling of the tongue was noticed by the medical attendant when she was two months old, and the mother thought the organ somewhat unnatural at birth. The part steadily enlarged, and when the patient was a year and a half old it began to protrude from the mouth. Upon examination the enlargement was found confined to the left half; the fungiform papillæ were increased in size; there was nowhere any vesiculation. A marked fulness, which could be made out to consist of knotted cords, was obvious in the submaxillary triangle and upper part of the neck. The anterior two-thirds of the enlarged left half of the organ were excised, and, at a later operation, the mass in the neck. Upon examination the enlargement of the tongue was found to be due solely to fibromatosis of the several nerves, which were enlarged throughout the organ, even in the conical and fungiform papillæ. The mass from the neck proved on dissection to be a typical plexiform neurofibroma; a portion of the submaxillary gland was removed with it, and this, too, was penetrated by certain of the diseased nerves, its lobules being in consequence unnaturally firm in consistence. The microscopic examination of the diseased nerves showed that their increase in size was due to a diffuse endoneurial growth of soft fibrous tissue.

In the discussion which followed the reading of the case, Dr. J. H. Thursfield related a similar one which had recently been under Mr. H. T. Butlin in St. Bartholomew's Hospital. A child, three years of age, was the subject of a swelling limited to the right side of the tongue, neck and face, which appeared to have commenced in the neck and to have afterwards spread to the tongue. The microscopical appearance of the portion of the tongue removed was also similar. (*Lancet*, 1902, ii., 1127.)

On March 4, 1903, Mr. Billington described to the Midland Medical Society a case of von Recklinghausen's disease in a man aged 38, who had died soon after the removal of a neurofibromatous tumour on his lingual nerve, which had come almost to fill the mouth. When three years of age, the patient had fallen and cut his cheek, two years after which a lump appeared, which increased in size. After partial removal at two operations, the right cheek remained much larger than the left. Later on lumps appeared in various parts of the body, many being subcutaneous. At the autopsy diffuse thickenings and fusiform swellings were found on the lumbar and sacral plexuses and their branches, and a tumour the size of an almond on the spinal cord. (*British Medical Journal*, 1903, ii., 671).

To this we may add two further references. One of these is a paper which was published by Rusca under the title "Contribucion al estudio de la macroglossia; caso de neurofibromatosis en el tronco y terminaciones del hipogloso." (*Rev. Ibero Am. de Cien. Med.*, Madrid, 1903, ix. 107.) The other is a contribution by Delfino (*Arch. per le Sc. Med.*, Torino, 1905, xxix. 34), who describes the condition in the tongue of a child and furnishes a good photograph of the organ and of the microscopic section.

The case now to be detailed occurred in a man, aged 24, who came under the care of Dr. George Gimmer, of Hounslow, for a swelling on the left side of the neck, which had been noticed for at least two years, and which latterly had been followed by an enlargement of the tongue on that side. In March, 1907, Dr. Gimmer removed from the left submaxillary region a tumour of the size of a small orange, having a naked-eye resemblance to a lipoma, and which was enucleated without encroaching upon the floor of the mouth. It had displaced, without involving, the submaxillary gland.

The report from the Clinical Research Laboratory states that the tumour resembled a neurofibroma, although no nerve-fibres had been detected. In the next three months the tongue had enlarged to more than double the size it was in March, and Dr. Gimmer wrote to Mr. Butlin, who passed the letter on to Mr. Spencer.

On the patient's admission into the Westminster Hospital, there was found a swelling strictly limited to the left half of the tongue, but continuous with a mass in the submaxillary region extending below the ala of the thyroid cartilage. The whole had a soft elastic or doughy consistence, and the skin over it was freely movable except in the line of the scar of the previous operation. The right half of the tongue was normal, but displaced to that side. Below the right angle of the mouth there was a pigmented molluscum fibrosum; there were no other indications of von Recklinghausen's disease. The patient could scarcely be understood in speaking; he had been reduced during the last three weeks to a liquid diet, and had begun to suffer from attacks of dyspnœa unless propped up at night.

On June 18, the whole mass, including the left half of the tongue and the floor of the mouth, was removed, together with the growth in the neck, through a submaxillary incision, along with a strip of skin, so as to include the scar of the former operation which had been performed by Dr. Gimmer. The growth was first isolated in the neck; it extended from the middle line beneath the deep fascia outwards to the carotid vessels, and downwards to the lower part of the anterior carotid triangle, within two inches of the sternum, the whole of the floor of the triangle, including the left ala of the thyroid cartilage, being exposed. The left half of the tongue was then separated from the right along the median raphe, a distinct layer of muscle still remaining between the raphe and the new growth. After cutting it away close in front of the hyoid bone and epiglottis, the right half of the organ was doubled back on itself and so fixed by sutures. The wound healed well and quickly. Before leaving the hospital the patient could eat and talk tolerably well.

PATHOLOGICAL ACCOUNT.

By S. G. Shattock.

The half of the tongue removed is, with the exception of its apex, abnormally firm in consistence, has lost its natural flexibility, and is considerably enlarged, as the following measurements will show. From the site of the foramen cæcum to the apex it measures in a straight line three inches and a quarter, and from the same site to the posterior limit an inch and a quarter. In thickness it attains a maximum of one and three-quarter inches.

Apart from its gross excess in bulk, there is nothing externally which gives any clue as to the nature of the enlargement. Many of the fungiform papillæ over the median area are abnormally prominent, but they are devoid of any appearance of vesiculation.

The median surface of the operation—that is, the surface from which the diseased half has been separated from the sound—presents centrally an extensive elevation of translucent, greyish aspect, and lobulated character; the surface itself is, however, smooth and intact, the morbid condition being accurately limited to the half of the tongue excised.

On dissection the lobulation of the eminence in question proves to be due to its consisting of a compact plexus of much enlarged nerves which, on the deep (*i.e.*, the lateral) aspect, penetrate and ramify through the muscular substance.

On making a sagittal section parallel with and slightly to the left of this surface, the whole of the muscular tissue is seen to be traversed by a dense plexus of enlarged greyish, semi-transparent nerves, the finer divisions of which can be traced in some situations up to the mucosa. (Pl., fig. 1.)

The disease is least pronounced in the apex, which, indeed, can hardly be said to participate in the enlargement, and retains its natural flexibility. The chief mass resulting from the neurofibromatosis occupies the inferior portion of the tongue, where the muscular tissue is so disparted and thinned out as to be hardly recognisable.

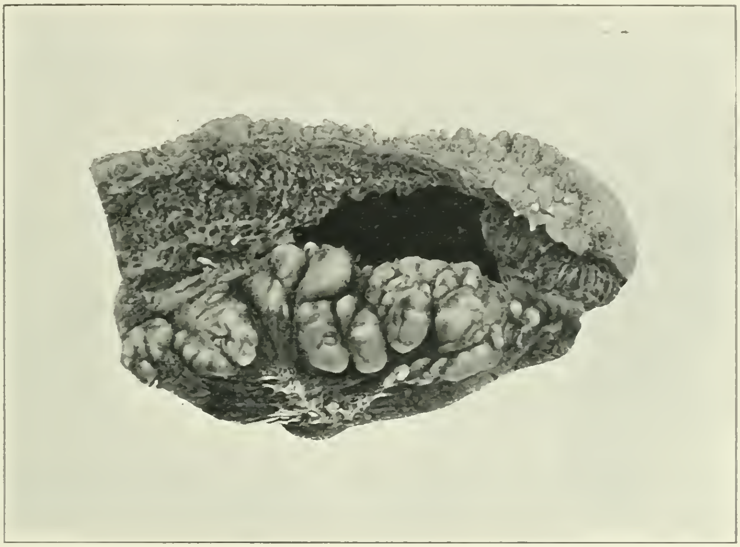

FIG. 3.

A thin slice removed from the inner aspect of the diseased half of the tongue excised, viewed from the plane of operation. From its centre there projects a well-defined lobulated mass of fibromatous nerves, the morbid condition being accurately limited by the mid-line. Natural size. (Mus. Royal College of Surgeons.)

The neurofibromatous plexus is distinctly circumscribed on the inferior aspect, as it is in the median surface of the removal, and is readily and cleanly exposed on stripping off the overlying sublingual gland, when it appears as a compact, slightly convex, lobulated mass, very like the salivary gland itself, except that its component parts are more firmly bound together, and are of denser consistence and greater transparency. That the mass in question is not salivary is proved by the complete absence of glandular tissue in microscopic sections; it consists solely of enlarged fibromatous nerves.

On the posterior surface of the removal the plexus is as well limited as on the others, the surface being formed, in fact, by an uncut and untorn collection of enlarged nerves, between which scarcely any other tissue is discernible.

On dissecting the parts removed in the operation, from the lateral aspect, the submaxillary gland is found to be displaced downwards by a close lobulated mass of enlarged nerves, which equals the gland in size, and lies in immediate contact with the upper surface of the latter, though easily separated by dissection from it. This mass projects immediately below the base of the tongue like a part of the submaxillary gland itself, but superiorly the enlarged nerves which compose it immediately penetrate the muscular substance to ramify through the organ. (Pl., fig. 2.)

On the inferior aspect the lingual nerve admitted of being displayed by dissection. Before their deeper penetration into the substance of the tongue its different branches are beset with small fibromata.

More posteriorly some of the larger subdivisions of the hypoglossal nerve are traceable beyond and into the muscular tissue. Some of these have a normal diameter; others are enlarged and misshapen from neurofibromatous disease.

In microscopic preparations the sections of groups of enlarged fibromatous nerves appear in the muscular substance and intermingled adipose tissue. The nerves themselves exhibit the changes to be presently described in the plexus from the neck.

From the corresponding side of the neck there was removed, in three parts, a large formation of the same kind as that connected with the tongue. The chief of these, on being lightly dissected, was resolved into strands of thickened tuberose nerves, six inches in length, although the dissection was by no means carried to the limit of freeing all the folds and tortuosities of the nerves composing it.

The other two portions removed at the operation were each, in the unravelled condition, about an inch and a half in diameter.

Histologically, the enlarged nerves from the neck consist throughout of a highly cellular, finely fibrillar connective tissue, in the midst of which, here and there, a nerve-fibre is recognisable.

The fibrillæ of the new growth evince no grouping in bundles, as in a firm fibroma, but interlace to produce a feltwork, the cells of which are equally devoid of any arrangement.

A certain number of the cells are highly vacuolated and of conspicuous size, corresponding with those named plasma cells by Schäfer, for the reason that the vacuolation of the cytoplasm results from the accumulation of clear fluid, presumably plasma.

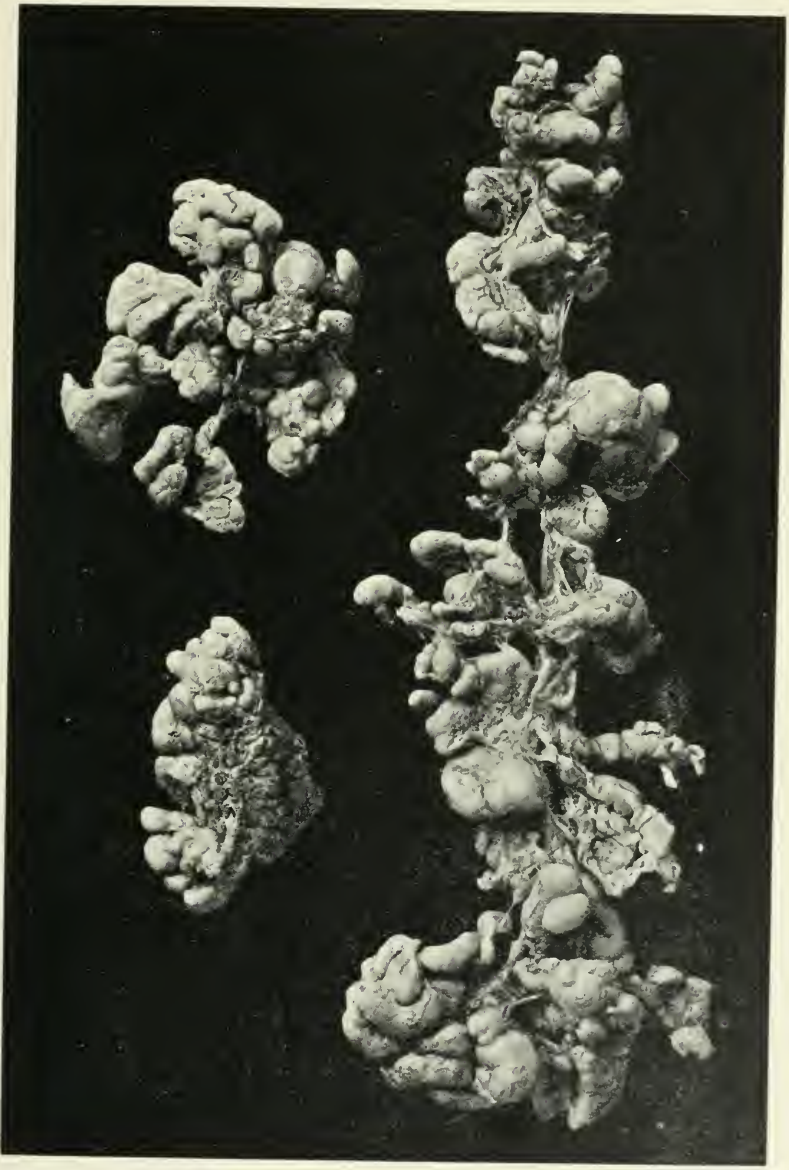

FIG. 4.

The three portions of the plexiform neurofibroma removed from the side of the neck. The enlarged nerves have been partially unravelled by dissection.

EXPLANATION OF PLATE.

Illustrating the communication upon a case of Macroglossia Neurofibromatosa.

By W. G. Spencer and S. G. Shattock.

Fig. 1.—A sagittal section of the diseased left half of the tongue removed by operation. The organ is much enlarged from neurofibromatous disease of its several nerves, sections of which are discernible in most of the divided surface. Below the muscular substance the enlarged nerves form a close plexus, the inferior free surface of which is distinctly circumscribed; superiorly the nerves of the "plexus" penetrate the substance of the tongue, through which they ramify. On the lower aspect of the specimen the lingual nerve is shown by dissection; its branches, before their deep penetration, are beset with small fibromata. More posteriorly there are shown, by dissection, some of the subdivisions of the hypoglossal nerve; certain of these are of normal size; others, thickened and misshapen from disease.

Fig. 2.—A dissection of the parts removed, viewed from the lateral aspect. Projecting immediately below the base of the tongue there is a prominent lobulated mass which at first suggests its being the submaxillary gland, but which is in reality a compact lobulated mass of enlarged and tortuous nerves which superiorly penetrate the muscular substance. Portion of the submaxillary salivary gland which lay below this mass, and which was removed at the operation, has been dissected away.

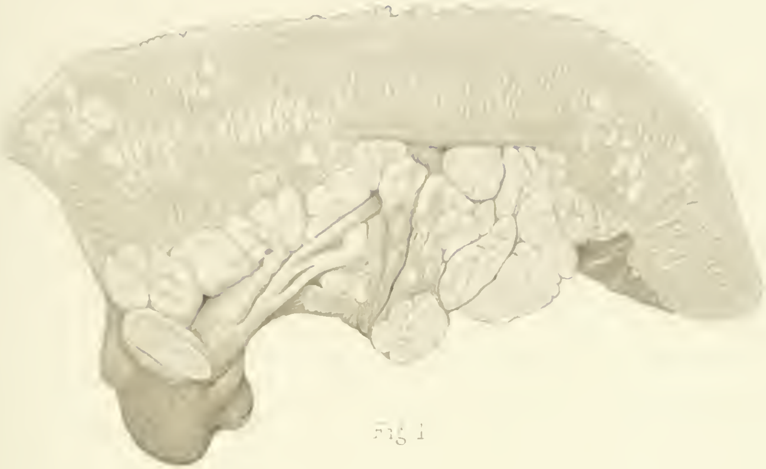

Fig 1

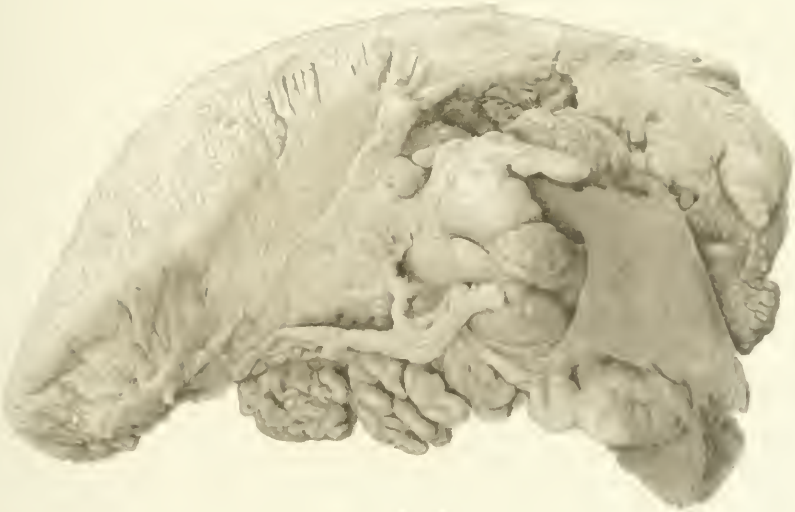

Fig 2

SPENCER and SHATTOCK: *Macroglossia Neurofibromatosa.*

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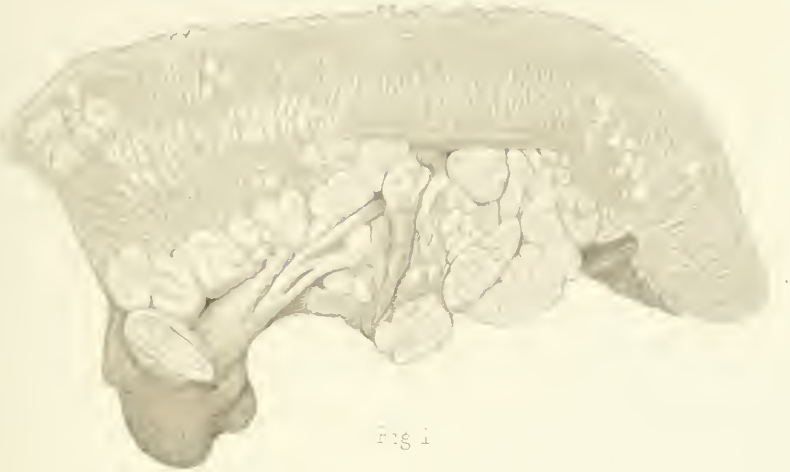

Fig 1

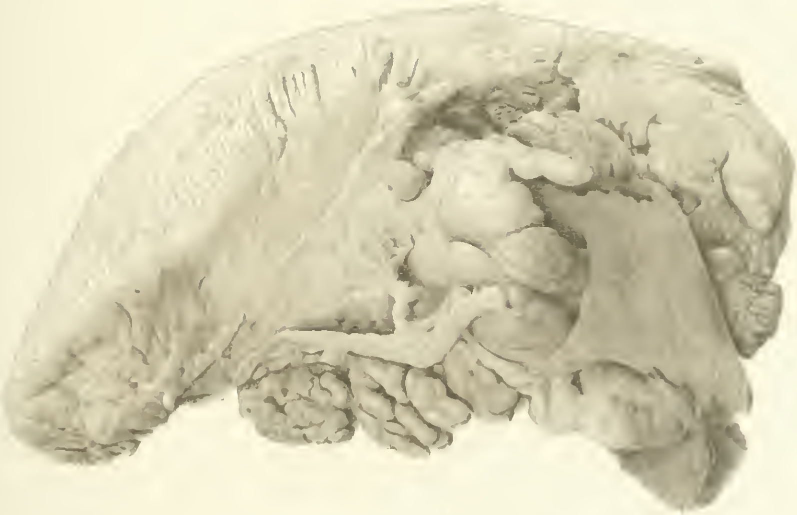

Fig 2

SPENCER and SHATTOCK: Macroglossia Neurofibromatosa.

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Pathological Section.

November 19, 1907.

Mr. S. G. Shattock, President of the Section, in the Chair.

A Contribution to the Study of the Relationship between Avian and Human Tuberculosis.

By S. G. Shattock, C. G. Seligmann, L. S. Dudgeon and P. N. Panton.

Synopsis.

(*a*) Introductory.
(*b*) The macroscopic forms of tuberculosis in birds.
(*c*) The feeding of pigeons with human tuberculous sputum.
(*d*) The inoculation of pigeons with human tuberculous sputum.
(*e*) The inoculation of pigeons with human tuberculous lymphatic glands.
(*f*) The inoculation of pigeons with the tuberculous organs of guinea-pigs infected with human sputum.
(*g*) The feeding of pigeons with the tuberculous organs of birds.
(*h*) The transference of ingested carbon to the mesenteric and Peyerian glands in the rabbit.
(*i*) The inoculation of pigeons with salt suspensions of the tuberculous organs of birds.
(*j*) The inoculation of guinea-pigs with salt suspensions of the tuberculous organs of birds.
(*k*) The feeding of a Rhesus monkey with the tuberculous organs of birds.
(*l*) Can the human tubercle bacillus be converted into the avian?
(*m*) The relation of the human to the avian bacillus as tested by means of the "opsonic" index.
(*n*) Conclusions.

The question raised by Koch in regard to the identity of bovine and human tuberculosis is one which cannot but be extended beyond these two particular forms of tubercular disease, and amongst others of kindred practical importance avian tuberculosis especially claims consideration. Although the relationship between the avian and human disease has been already debated and investigated (as was that between the bovine and human forms before it was made the subject of a Royal Commission), it will, we submit, bear reopening, and this partly because methods of investigation of more recent origin than those hitherto adopted may be applied in its elucidation.

The extensive consumption of birds as articles of human food invests the subject with a large degree of practical importance, and correlated with this is the question how far avian tuberculosis may be contracted from a human source. In our investigation we have included a method of testing the two bacilli which has not before been practised in this connection, viz., a study of the phagocytosis of the two micro-organisms (the avian and human) when presented to the normal polymorphonuclear leucocytes in the presence of tuberculous serum which has first been saturated with one of the bacilli in question. Just as in the case of the agglutination test, the phenomenon, although first employed in the diagnosis of a microbial disease, may be conversely applied to the diagnosis of a bacillus, so may the opsonic phenomenon be used in attempting to determine not only the specific character of a serum, but, conversely, the specific character of a bacillus.

The problem which presents itself in the case of avian and human tuberculosis may be best stated in the form of questions:—

(1) Is the disease the same?

(2) Is the disease the same, but modified in form?

(3) Are there two distinct diseases?

The answers to these questions depend upon those which can be given if the term "bacillus" is substituted for "disease." The question resolves itself into the identity or non-identity of the bacilli producing the lesions. We must class as modified forms of the same disease all which are caused by the same bacillus; whereas, on the contrary, diseases produced by different bacilli, though they may from the standpoint of morbid anatomy be alike, should receive different names.

As, in the case of the human disease, a point of departure is obtained by taking a typical example of pulmonary tuberculosis, so, in that of the avian, a "typical" case may be taken as one in which, with or without ulceration of the intestine, there is tuberculosis of the spleen and liver, the lungs being uninvolved. This may be selected as a standard of the avian disease, since it is the usual form which fatal tuberculosis assumes in birds. We may in this place recount the other forms that may be met with, and for this purpose we have utilised the data furnished by the examination of birds dying during the last two years from tuberculosis in the Gardens of the Zoological Society. During this period autopsies have been made upon about 500 birds, of which about 150 presented lesions referable to tuberculosis.[1]

The most common form of tuberculosis occurring in the Gardens is that which general experience shows to be the most usual in other localities. By far the largest number of birds present the abdominal form of the disease. In a typical example the liver and spleen are crammed with white or yellowish nodules; in the spleen these may be so abundant that little or no trace of the proper splenic substance may remain. The mass representing the spleen is in most cases considerably larger than the organ it replaces, firm, and more or less gritty on section. Tubercular lesions of the spleen in birds never seem to attain the horn-like density and keratinous appearance which are common in advanced tubercular lesions of the liver. Tubercular lesions in the liver may, even when the disease is advanced, remain small, the organ being speckled with innumerable whitish foci, individually not larger than a pin's head; more frequently the liver contains a smaller number of tubercular foci of larger size. It is uncommon to find confluent masses, the individual foci being not larger, as a rule, than marrow-fat peas. Such nodules are often remarkably tough, and present a hyaline, keratinous appearance; they are seldom, if ever, extensively calcified.

There may be complete absence of any sign of intestinal disease in cases which present the advanced lesions just noticed; or there may be a slight general or patchy redness of the small intestine, with nothing to show that this slight enteritis is tubercular; or there may be a few enlarged lymphatic follicles in the gut, in which microscopic examination shows an abundance of tubercle bacilli. Ulceration is uncommon. Although this is so, tubercular enlargement of the mesenteric glands is not rare, and this often in association with lesions of the spleen and liver. Apart altogether from intestinal ulceration, there may be considerable enlargement of the lymphoid follicles, so that nodules result, over which the villi may themselves be enlarged, a remarkable papillary condition of the mucosa being so produced. Sometimes the whole of the large gut is thickly covered with these wart-like upgrowths, which on microscopic section are found to contain dense masses of acid-fast bacilli; at other times the whole of the small gut may be so affected. In one such instance (that of a Burmese slaty-headed paraquect, *Palæornis* sp.) there was no macroscopic disease in any other of the abdominal viscera than the intestine, and none in the thorax.

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[1] The parrot family is not here considered. Tuberculosis is rare in members of this group of birds kept in the Zoological Gardens.

Cases occur, by no means rarely, in which, associated with lesions of the abdominal viscera, there is advanced disease of the lung, both organs being affected. The pulmonary disease in such circumstances does not appear to arise *ex contiguo*, but to be due to the appearance of new foci.

The lesions are much drier than those occasioned by the *Bacillus tuberculosis* in the human or simian lung. They present themselves, usually, as dry masses continuous with the pulmonary parenchyma around, without the formation of a limiting capsule. The consistence of such foci may be fairly firm, and they may be more or less calcified. In colour they vary from white to a pronounced yellow, and are often mottled with the pigment previously deposited in the pulmonary tissue. Neither the formation of pus nor cavitation has been observed.

Other organs in the higher part of the body are rarely affected. On one occasion the thyroid glands of a peafowl were found enlarged to the size of a filbert, tough, and calcareous. In this case the organs affected were diagnosed rather by their site (one on either side, at the root of the neck) than by means of the microscopic sections made of them, so altered were the glands in structure. The intestines of this bird, together with the liver and spleen, were extensively diseased, as were likewise the lungs.

As another unusual location, the thymus may be involved. In a roller (*Coracias garrulus*), the intestine of which presented numerous minute tubercles without any ulceration, and in which the spleen and liver were extensively diseased, there was on the left side an enlargement of the thymus (which in birds extends the entire length of the neck), the diseased structure taking the appearance of a chain of tubercular lymphatic glands. Microscopic examination of portion of this proved the tissue to be full of acid-fast bacilli.

At times a tubercular mass in the lung will be continuous with one outside the ribs situated beneath the deep fascia. But besides subcutaneous masses so continuous with lesions within the thorax, diffuse tubercular formations at times occur beneath the skin, or between the muscles, apart from direct extension from other affected sites. Intramuscular masses of this kind may reach a considerable size, even in

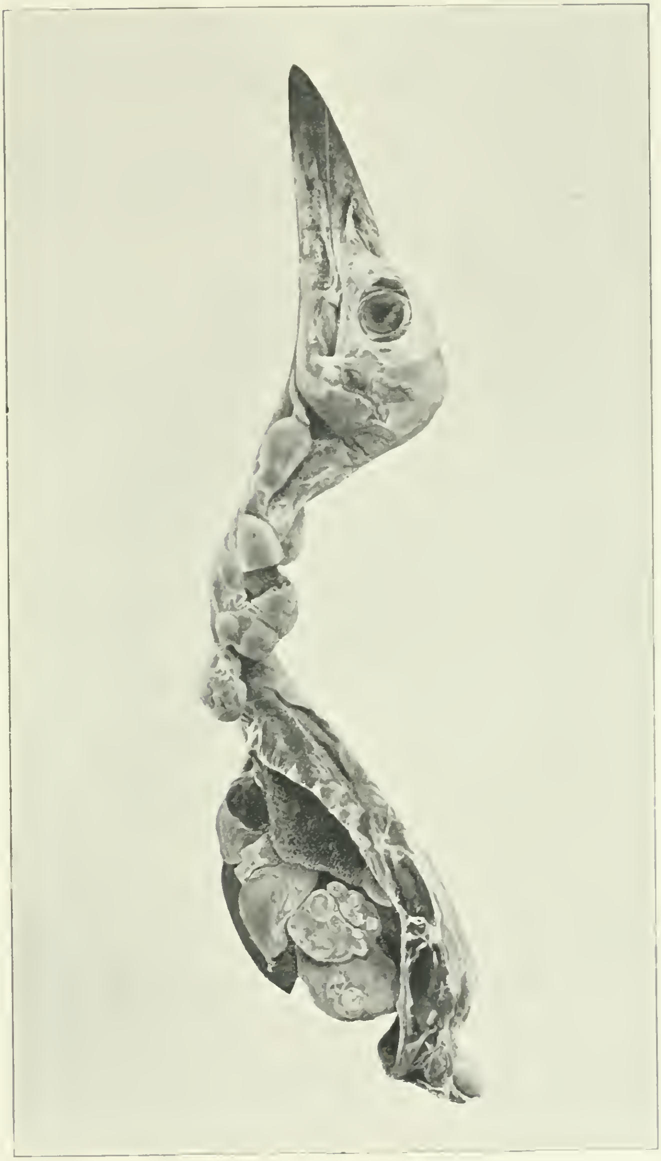

FIG. 1.

A Roller *(Coracias garrulus)* showing on the left side an enlargement of the thymus gland (which gland in the bird normally extends the entire length of the neck), the diseased structure taking the appearance of a chain of tubercular lymphatic glands. The spleen and liver are diseased, the lungs unaffected. (4911 B. Mus. Royal College of Surgeons. Natural size.)

small birds. A lark presented three nodules beneath the skin, none of them smaller than a pea, and unconnected with the deep structures. Microscopic examination of the nodules showed an abundance of acid-fast bacilli. Irregular plaques of a peculiarly white colour are at times encountered in the intermuscular spaces.

There is one form of abdomino-thoracic tuberculosis in birds the origin of which is obscure. The viscera, abdominal and thoracic, are matted into a more or less solid mass, but are not themselves, as a rule, adherent to the body-wall. The liver is invariably much enlarged and projects downwards, sometimes almost to the vent; no nodules are to be seen in the liver, although smears or sections display large numbers of bacilli in its tissue. There are no nodules visible on the intestine, the coils of which are everywhere adherent to each other and to the solid viscera.

It is noteworthy in regard to avian tuberculosis that, speaking broadly, the kidneys are not affected. The only exception we have encountered is that of a pigeon injected intraperitoneally with a suspension of the avian bacillus. In this bird marked peritoneal tuberculosis was set up, one of the kidneys being involved by direct extension.

It is perhaps necessary to refer to the co-existence of tuberculosis and aspergillosis in birds. Aspergillosis is a disease from which water-fowl especially suffer, though it is by no means limited to such. In some cases of aspergillosis a guinea-pig, inoculated from what appeared to be typical lesions of this condition, subsequently developed tuberculosis of the characteristic local type that is produced by inoculating this animal with avian tubercle. In cases of this kind the original bacillus could not commonly be detected even after prolonged microscopical search, though occasionally it was found in small numbers, especially if an emulsion of the wall of the air-sac was made with white of egg, and smears prepared from the thick fluid so produced. In the majority of cases the mould which causes the lesions can be seen growing within the thickened air-sacs, although where other serous membranes, like the pericardium and the bounding layers of connective tissue of the mediastina are affected, the aspergillus may not be obvious. In a few cases the only micro-organism discoverable by examination of the air-sacs, presenting the thickened yellowish exudate suggestive of aspergillosis, was an acid-fast and presumably tubercle bacillus.

As an illustration of this statement the post-mortem examination of a bell-bird (*Chasmorynchus* sp.) may be cited. The lungs, liver, spleen, intestines and kidneys appeared normal; a stiff yellowish substance formed a box-like chamber in the anterior mediastinum, through which the trachea ran. The great vessels at the base of the heart were also covered with similar material, which did not invest the pericardium, but ran down, encasing the œsophagus, into the abdomen. On the abdominal surface of the lungs, the walls of the anterior abdominal air-sacs were so thickened as to be quite stiff, and on opening them they exhibited a definite slit-like patulous lumen; the walls were nowhere gritty, and repeated microscopic examination of smears made from their moist inner surface showed no micro-organisms of any kind; it was only when a portion was crushed up with white of egg, and smears made from this, that sparse acid-fast bacilli were discovered.

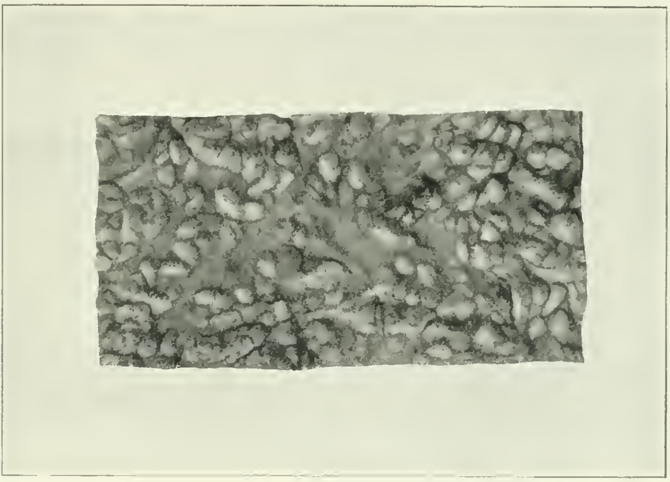

Fig. 2.

Portion of the intestine of a Mantchurian crossoptilon, showing a subpolypoid condition suggestive of a tubercular infection, but due to the presence of an animal parasite in the intestinal wall. (Mus. Royal College of Surgeons. Natural size.)

As in the case of mammals, so in birds, lesions produced by the higher animal parasites may be so localised and multiple as to suggest tuberculosis. In the lungs of horses, dogs, cats, and especially of sheep, nodules due to the presence of the ova or larvæ of nematode worms ("Wurm-Knötchen") are well known as simulants of true tuberculosis.

In one bird (Mantchurian crossoptilon) the intestine showed a curious subpolypoid condition over a considerable length, highly suggestive of a tubercular infection. The lesions proved, however, to be due solely to the presence of minute round worms in the wall of the bowel, and no acid-fast bacilli were demonstrated in them by the usual method. In this bird the liver and spleen were full of "tubercles," but smears from these showed large numbers of acid-fast bacilli. The bird in this case was thus affected with two distinct diseases.

It is chiefly amongst birds kept in captivity that tuberculosis is met with. Although the disease is so rife in the Gardens of the Zoological Society, in the case of ten wood-pigeons shot whilst visiting the Gardens no tuberculosis was found. So rare is the disease amongst wild birds that we venture to append a figure showing advanced tuberculosis in a lapwing which was captured in a moribund condition in Scotland. For this bird we are indebted to Mr. H. S. Gladstone.

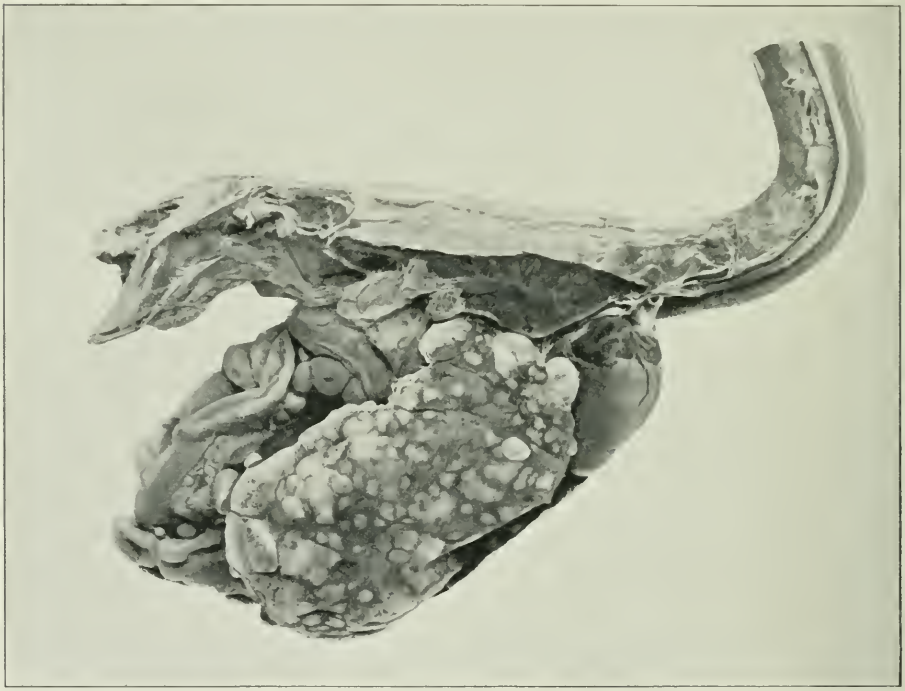

Fig. 3.

A lapwing *(Vanellus vulgaris)* which was captured in a moribund state and is the subject of advanced tuberculosis, the liver being much enlarged and thickly strewn with tubercles. Tuberculous nodules of varying size are scattered over the peritoneal surface of the intestines and on the peritoneum elsewhere. The lungs are quite free of disease. The hepatic lesions showed microscopically vast numbers of acid-fast bacilli. (4911 A. Mus. Royal College of Surgeons. Natural size.)

Reverting to the common or typical form, then, of avian tuberculosis, viz., that arising by way of the intestine, it is still a common belief that the disease is contracted from a human source, from the ingestion of sputum expectorated by the phthisical.

At the outset, this important question has to be determined: Can tuberculosis be induced in birds by means of human tuberculous sputum?

THE FEEDING OF PIGEONS WITH TUBERCULOUS HUMAN SPUTUM.

First Group of Experiments.

The pigeons were fed with bread made into a thick paste with sputum.[1] The sputum was derived from cases of pulmonary tuberculosis, in each of which the presence of tubercle bacilli was proved by microscopic examination, and in some, in addition, by the injection of guinea-pigs. The paste was given three times a week, each bird receiving three portions, the bolus being of a size that could be conveniently held in a pair of forceps. Between times the birds were kept on soaked bread only, without any kind of grain. No grit was allowed.

Pigeon 1.—Fed for four weeks.

Post Mortem.—Wasted. Intestines congested. No macroscopic evidence of tuberculosis in the viscera. Microscopic examination of the gut, at three different levels, and of the spleen, showed no tuberculosis and no tubercle bacilli.

Pigeon 2.—Fed for seven weeks.

Post Mortem.—Wasted. Viscera normal to naked eye. Microscopic examination of the intestines showed no lesions and no tubercle bacilli. The excrement of this bird was examined microscopically on three occasions; on the first a single tubercle bacillus was encountered; on the second a few; and on the last large numbers.

Pigeon 3.—Fed for eleven weeks.

Post Mortem.—Little wasting. Viscera, including intestines, normal. Spleen slightly larger than natural. A salt suspension was made of the spleen, and 2 c.cm. injected into the leg of a guinea-pig. The animal, on being killed twelve weeks later, presented no signs of tuberculosis.

Examination of the excrement on two occasions showed a few tubercle bacilli.

Summary of the Results of the preceding Group of Experiments.

Three pigeons were fed for four, seven, and eleven weeks respectively upon bread alone, with which tuberculous sputum had been mixed at regular intervals. None contracted tuberculosis.

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[1] The birds used for the whole of our experiments have been healthy pigeons, none amongst which ever developed spontaneous tuberculosis.

THE FEEDING OF PIGEONS WITH TUBERCULOUS HUMAN SPUTUM.

Second Group of Experiments.

In these, bread made into a paste with sputum was given three times weekly, each bird receiving three portions, as in the preceding experiments; but beyond this the birds were fed with grain in the usual way.

Pigeon 1.—Fed for seven weeks.

Post Mortem.—Well nourished. No visceral disease. Microscopic examination of the intestine showed no lesions and no tubercle bacilli.

Pigeon 2.—Fed for eight weeks.

Post Mortem.—Well nourished. Viscera normal. Microscopic examination of the intestine at three levels showed no tubercle bacilli.

Pigeon 3.—Fed for eleven weeks.

Post Mortem.—Well nourished. In the small intestine there was a patch of congestion in which the solitary glands were obvious; numerous round worms lay in the gut. Viscera normal. Microscopic sections carried through the congested area showed a chronic inflammatory condition, but no tubercular lesions. Microscopic examination of the excrement on two occasions showed, on one, the presence of tubercle bacilli, and on the other none. A salt suspension of the spleen was injected subcutaneously into a guinea-pig. The animal died two weeks later.

Post Mortem.—There was no nodule at the site of injection, and no visceral tuberculosis.

Pigeon 4.—Fed for twelve weeks.

Post Mortem.—Well nourished. No visceral tuberculosis. Microscopic examination of the intestines at two levels showed no tubercle bacilli. The excrement of this bird and of No. 2 (which were kept together) showed no tubercle bacilli on any of the six occasions on which it was examined. The spleen of one of these two birds was examined for tubercle bacilli, with a negative result. The sputum used for these two birds proved highly pathogenic to guinea-pigs.

It is noteworthy, in passing, that the tubercle bacillus was found in the excrement of those birds which had been fed with bread and sputum alone, without grain (grit also being excluded), on every occasion on which it was looked for. In contrast with this result, the excrement of two of the birds (kept together) which had grain in addition to the bread paste and sputum showed no tubercle bacilli on any of the six occasions on which it was examined. The uniformity of the contrast, however, is marred by the circumstance that in the case of one bird, fed for eleven weeks on bread and grain, the excrement examined on two occasions showed on one no bacilli, but many on the other.

Summary of the Results of the preceding Group of Experiments.

Four pigeons were fed for periods of seven, eight, eleven, and twelve weeks with tuberculous sputum made into a paste with bread, and also with ordinary grain. None contracted tuberculosis.

THE FEEDING OF PIGEONS WITH TUBERCULOUS HUMAN SPUTUM.

Third Group of Experiments.

Eight pigeons were fed in the following manner: The sputum was supplied regularly from typical cases of pulmonary tuberculosis, and in each case the presence of tubercle bacilli was demonstrated by microscopic examination. It was forwarded every other day in bottles furnished with rubber stoppers, and kept in the cold. Some of the sputum being transferred to a glass dish, a series of peas were thrown in and stirred with a glass rod. The mouth of each bird was then held open, and five of the peas, with adhering sputum, were dropped in succession into it by means of a long pair of forceps. This procedure was carried out almost daily with the exception of Sundays. The birds were kept in zinc cages with removable bottoms, which were regularly cleaned to prevent the possible drying of bacilli in the excreta, and the occurrence of air-borne pulmonary infection.

Pigeon 1.—Fed six weeks.

Post Mortem.—Well nourished. All the viscera healthy, including the intestines, which were slit up and carefully examined, as in all the birds which follow.

Pigeon 2.—Fed thirteen weeks.

Post Mortem.—Well nourished. Viscera all normal.

Pigeon 3.—Fed nineteen weeks.

Post Mortem.—Well nourished. Viscera healthy; no lesions of the intestine.

Pigeon 4.—Fed nineteen weeks; well nourished.

Post Mortem.—Liver, kidneys, lungs: nothing abnormal. The intestine, about 8 inches below the gizzard, showed some patchy congestion, and minute whitish elevations on the mucosal aspect; no ulcers. A few inches further on there was a second similar area. Microscopic examination of the suspicious areas showed no tubercular lesions and no tubercle bacilli. The spleen in section showed the Malpighian corpuscles particularly plainly. Microscopic examination showed no tubercular foci. The chief part of the spleen was used to make a salt suspension, the whole of which (1 c.cm.) was injected beneath the skin of the thigh of a guinea-pig. No local nodule had formed and no enlargement of inguinal glands had occurred thirty-two days after the injection. The changes present, therefore, must be ascribed to an enteritis set up by other micro-organisms in the sputum than the tubercle bacillus.

Pigeon 5.—Fed nineteen weeks.

Post Mortem.—Nutrition moderate. Liver, kidney, lungs: normal. Intestine: patchy congestion irregularly distributed, most pronounced about 14 inches below gizzard. Minute whitish elevations (solitary glands) occur in the congested areas; no peritoneal tubercles. No ulcers occur. Microscopic examination shows no tubercular disease. The spleen (which presented no histological evidence of tuberculosis in sections) was rubbed with salt solution in a mortar, and 1 c.cm. of the suspension injected beneath the skin of a guinea-pig. A nodule had formed at the site of injection eleven days later; the inguinal glands were felt to be enlarged thirty-two days after the date of the injection. The animal died nine weeks after the experiment.

Post Mortem.—Small ulcer at site of inoculation; inguinal glands much enlarged; smears of one of the glands showed beaded acid-fast bacilli. Lumbar glands of the corresponding side enlarged. Spleen enlarged with prominent groups of miliary tubercles. Microscopic examination showed the presence of tubercle bacilli. One grey tubercle in each lung. Nothing apparent in the liver to the naked eye either on surface or in section.

Pigeon 6.—Fed thirty-three weeks.

Post Mortem.—Liver, lungs: normal; spleen: small, normal. Intestines: no tubercles on outer aspect, no ulcers of mucosa; some patchy congestion accompanied with abnormally conspicuous solitary glands. Microscopic examination of the gut and spleen showed no tuberculosis.

Pigeon 7.—Fed thirty-five weeks.

Post Mortem.—All viscera normal. No ulcers of intestine; no enlargement of solitary glands.

Spleen made into a suspension with salt solution and injected into the subcutaneous tissue of the thigh of two guinea-pigs, $\frac{3}{4}$ c.cm. being injected into each. One of the animals was killed twenty-eight days later; no trace of disease at site of injection; no enlargement of the inguinal glands; abdominal viscera, quite normal. The other guinea-pig was killed fifty-one days after the inoculation. *Post mortem:* All the viscera and the site of inoculation quite healthy.

Pigeon 8.—Fed thirty-six weeks.

Post Mortem.—Bird in excellent condition. Liver, lungs, spleen: normal. Intestines: no trace of disease. The spleen was used to make a suspension in salt solution, the whole of the suspension (1 c.cm.) being injected beneath the skin of the thigh of a guinea-pig. The animal was killed fifty-nine days later. It was found to be absolutely healthy throughout.[1]

Summary of the Results of the preceding Group of Experiments.

In none of these eight pigeons, fed almost daily for periods varying from six to thirty-six weeks with human sputum and grain, did any intestinal tuberculosis develop, or any disease of the abdominal or thoracic viscera.

Five guinea-pigs were injected subcutaneously with a salt suspension of the spleens of four of the birds. No disease was induced in the guinea-pigs, except in one instance.

Negative results were obtained in the case of birds fed for nineteen weeks, thirty-five weeks (two guinea-pigs injected), and thirty-six weeks. The positive result came from a bird fed nineteen weeks, the spleen of which showed no macroscopic disease. The guinea-pig in this case developed typical tuberculosis of the form set up in this animal by the injection of the human bacillus. There was no flaw in the injection itself; no leakage occurred, and the site of puncture was sealed with collodion.

It may be assumed that in this bird bacilli were transferred to the spleen through an intact intestinal mucosa, without producing any lesion; that they were deposited as innocuous particulate material might be, and that when transplanted into the suitable tissue of the guinea-pig these human bacilli produced their ordinary pathogenic results. A series of similar results which followed the intramuscular injection of sputum in pigeons, when the injection of the macroscopically normal spleen of the birds produced typical generalised tuberculosis in guinea-pigs, is recounted towards the end of the communication.

It thus appears that the pigeon cannot be infected with tuberculosis by means of phthisical sputum. As an interesting pendent to these results, which show that the source of avian tuberculosis is not the ingestion of such sputum, we may adduce certain information furnished to us by Mr. R. I. Pocock, the Superintendent of the Zoological Society's Gardens.[1]

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[1] In the case of this group of experiments, four other birds from the same source were kept in the country as controls; these were killed two, three, and six months afterwards, and found to be perfectly healthy.

In spite of the absence of phthisis among the keepers of the aviaries, however, tuberculosis of the intestino-abdominal type is a very common cause of death amongst the birds kept in the Gardens.

THE INOCULATION OF PIGEONS WITH TUBERCULOUS HUMAN SPUTUM.

In these experiments, sputum derived from cases of pulmonary tuberculosis was made into an emulsion with sterilised normal salt solution, and injected into pigeons. The spots selected were in some cases the base of the wing, and in others the leg. With the first portion injected the skin became raised; the needle was then pushed more deeply into the muscle itself. In many instances the result was controlled by the injection of portions of the same material into guinea-pigs. Six birds were thus injected, with the following results:—

Pigeons 1, 2, 3 were injected in the subcutaneous tissues and muscles of the leg with 1 c.cm. of a thick saline emulsion of sputum. They were killed after ten weeks.

Post Mortem.—In two an abscess formed at the site of injection; the neighbouring subcutaneous gland had broken down into a thick pultaceous material. The viscera were normal. No tubercle bacilli were found in the pus from the abscess. The spleen from one of the birds was examined microscopically, and found to be normal. In the third bird the site of injection was healthy, and there was no evidence of disease elsewhere.

Three guinea-pigs were injected subcutaneously with 4 c.cm. of a similar emulsion of the sputum from the same patient. Two of the animals died of sepsis a week later, without any evidence of tuberculosis. The third guinea-pig became ill, and was killed a month after the injection.

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[1] Mr. Pocock has been good enough to ascertain how many cases of phthisis have occurred amongst the keepers within recent years. He writes as follows:—"The enquiries I have made as to the prevalence of consumption amongst the keepers in the Zoological Gardens show that this disease is almost unknown. There is only one case on record, viz., that of Henry Webb, a native of Southampton, who entered the Society's service as keeper in the antelope house in 1883, retired from ill-health in 1903, and died from consumption the same year. He has been described to me as consumptive in appearance on his arrival. With regard to keepers in the monkey house, Charles Richardson, appointed about 1860, retired from ill-health at the end of 1896, and died in April of the following year. Consumption was not the cause of his death, so far as I can ascertain. Eustace Jungblut, appointed in 1886, retired in 1902 and is still living. The men who succeeded the above-mentioned keepers in the antelope and monkey houses are still in the Society's service, and are apparently in perfect health."

Post Mortem.—Local abscess; glandular enlargement. Smears from a breaking-down gland showed a large number of tubercle bacilli. Tubercles were present in the spleen and liver.

Pigeon 4.—Injection made into the base of the wing with 1 c.cm. of a thin emulsion of sputum from a young man with a three months history of acute pulmonary tuberculosis. The bird was killed after eleven weeks.

Post Mortem.—Bird well nourished; no evidence of disease anywhere found; no local nodule. Two guinea-pigs were at the same date injected subcutaneously with a similar amount of the same emulsion of sputum. Both became ill, and were killed six weeks after the injection.

Post Mortem.—In each there was a local caseous abscess, and extensive tuberculosis of lymphatic glands, spleen, liver, lungs, heart, and mediastinal glands.

Pigeons 5, 6.—Into the base of the wing were injected 2 c.cm. of a thin salt suspension of tubercular sputum. Killed after seven weeks. In each a small patch of necrotic material was found lying superficially on the pectoral muscle at the site of inoculation. Tubercle bacilli were present in smears made from the necrotic substance. Smears from the spleens of both birds, which presented no macroscopic marks of disease, showed no tubercle bacilli. A salt suspension of the spleen of one of the pigeons was injected subcutaneously into a guinea-pig: the animal was killed five weeks later, and showed no evidence of disease. A salt suspension of the necrotic patch was injected subcutaneously into another guinea-pig. The animal was killed five weeks later.

Post Mortem.—Tuberculosis of popliteal, inguinal, and lumbar glands; spleen and liver not affected. A salt suspension of the tuberculous lumbar gland of this guinea-pig was injected subcutaneously into another guinea-pig. The animal died nine weeks later.

Post Mortem.—Animal wasted; small local caseous abscess; tuberculosis of lymphatic glands, extending upwards into the axilla and neck; spleen and liver enlarged, with caseous areas; several tubercles on the lungs. A guinea-pig was injected with the same amount of the same emulsion of the sputum used to inoculate these two pigeons, Nos. 5 and 6. The animal was killed three and a half weeks later.

Post Mortem.—Local abscess, tuberculosis of lymphatic glands, spleen, and liver. Smears from the spleen showed numerous tubercle bacilli.

Summary of the Results of the foregoing Group of Experiments.

Of six pigeons injected with a salt emulsion of tubercular sputum, two showed no lesion (ten and eleven weeks respectively); two showed local sepsis, but no tuberculosis (ten weeks); two showed a slight local lesion (seven weeks).

Of six guinea-pigs similarly injected, two died from sepsis (one week); four showed advanced tuberculosis (three and a half, four, and six weeks).

In the case of one pigeon which exhibited a local lesion (seven weeks), a suspension made from the lesion produced a limited tuberculosis in a guinea-pig in five weeks; this, on being carried into a second guinea-pig, produced marked tuberculosis and death in nine weeks. A suspension of the spleen of the same pigeon gave a negative result when injected into another guinea-pig.

THE INOCULATION OF PIGEONS WITH A SALT SUSPENSION OF TUBERCULOUS LYMPHATIC GLANDS FROM THE HUMAN SUBJECT.

Pigeon 1.—Into the base of the wing were injected 2 c.cm. of a salt suspension made from a tuberculous gland removed from the human axilla. Histologically the gland showed the typical marks of tuberculosis; one tubercle bacillus was encountered. There was no clinical evidence of disease elsewhere. Killed after six weeks.

Post Mortem.—No local nodule; nowhere any evidence of disease.

Pigeon 2.—Into the base of the wing a salt suspension made from a tuberculous gland from the human neck was injected. Histologically the gland presented the typical marks of tuberculosis. There was no clinical evidence of disease elsewhere. Killed after four weeks.

Post Mortem.—No local or other disease.

Summary of the Results of the foregoing Group of Experiments.

A salt suspension of tuberculous human lymphatic glands (axilla and neck), from two different patients, produced no disease in either of two pigeons on subcutaneous and intramuscular injection.

THE INOCULATION OF PIGEONS WITH THE TUBERCULOUS ORGANS OF GUINEA-PIGS INFECTED BY MEANS OF HUMAN SPUTUM.

In this series human sputum was first injected into guinea-pigs, and suspensions afterwards prepared from their tuberculous organs for the inoculation of pigeons, with the object of eliminating for the birds the septic element in the sputum, and at the same time of contrasting the relative pathogenicity of the same strain of human bacillus for the guinea-pig and pigeon. Fourteen pigeons were thus injected.

Experiment A.—Of a thick emulsion of tuberculous sputum 4 c.cm. were injected subcutaneously into a guinea-pig. The animal was killed four weeks later. *Post Mortem :* Advanced tuberculosis, the liver being also involved. A salt suspension was made of the spleen. Of this, 1 c.cm. was injected into the base of the wing of each of two pigeons. The two birds were killed nine weeks later. They were in good condition ; no local nodule, and no disease in any of the organs was found. On the same day as that on which the pigeons were injected 1 c.cm. of the same splenic suspension was injected subcutaneously into each of two guinea-pigs. One of these animals died in a week. *Post Mortem :* No evidence of sepsis or of tuberculosis. The other animal was killed six weeks later, and was found to have advanced tuberculosis, the lungs being also involved. A splenic suspension was made from this guinea-pig, and of it 2 c.cm. were injected subcutaneously into a pigeon (3). This bird was killed six and a half weeks later. *Post Mortem :* At the site of inoculation there was found a small necrotic patch which could be enucleated from the surrounding tissues. Smears from the necrotic substance showed numbers of tubercle bacilli, and very few cells. No internal disease was found. A suspension made from this necrotic material was injected subcutaneously into a pigeon. This bird was killed eight and a half weeks later. *Post Mortem :* No local lesion ; no disease of the viscera.

Experiment B.—Of a thin emulsion of tuberculous sputum 1 c.cm. was injected subcutaneously into each of two guinea-pigs. Six weeks later the animals were killed ; they were found to have advanced tuberculosis, the lungs of one being extensively involved in addition to the other organs. A salt suspension of the lung (2 c.cm.) was injected into the base of 'the wing of a pigeon (4). A salt suspension of the spleen (2 c.cm.) of the other tuberculous guinea-pig was injected into the base of the wing of another pigeon (5). Both these birds were killed eight and a half weeks later. *Post Mortem :* Both were in good condition ; no local nodule had formed, and there were no lesions in the viscera.

Experiment C.—Two guinea-pigs were inoculated in the subcutaneous tissue of the thigh with tubercular sputum. Each developed an ulcer at the site of inoculation, caseous enlargement of the inguinal glands, and tuberculosis of the spleen. Thirty-eight days later one of the animals was killed and its spleen used to make a salt suspension. Of this suspension 1 c.cm. was injected into the substance of the pectoral muscle of each of three pigeons (6, 7, 8). The injection was entirely intramuscular; collodion was dropped over the puncture in the skin on the withdrawal of the needle. No local swelling was at any time felt at the site of experiment. One of the birds was killed six weeks later; the other two were killed fourteen weeks after the inoculation. In none was there any local lesion found in the pectoral muscle, or any disease of the viscera.

Experiment D.—A guinea-pig was injected subcutaneously with 2 c.cm. of a thin emulsion of tuberculous sputum. The animal died in fourteen days. *Post Mortem:* Local caseous abscess; tuberculosis of popliteal and iliac glands; spleen and liver normal. Of the tuberculous glands a salt suspension was made and used as an injection; into the base of the wing of each of two pigeons (9, 10) 2 c.cm. of this suspension was injected. One pigeon was killed after three days. *Post Mortem:* Nothing was found at the site of inoculation, and no disease elsewhere. The spleen was normal. Of the spleen a saline suspension was made and injected into the leg of a guinea-pig; the animal died after six weeks. *Post Mortem:* Local caseous abscess, tuberculosis of lymphatic glands and of the spleen and liver; smears from the abscess showed large numbers of tubercle bacilli; sections of the spleen showed typical tubercular lesions containing acid-fast bacilli. The other pigeon was killed eight days after the injection into the base of the wing. *Post Mortem:* A small abscess with indurated margin was found at the site of inoculation, in the muscle substance; viscera healthy; smears from the abscess showed lymphocytes and a few tubercle bacilli; sections made through the wall of the abscess showed no giant-cells and no caseation, but extensive areas of lymphocytic infiltration.

Experiment E.—A guinea-pig was inoculated with 2 c.cm. of a thin saline emulsion of tuberculous sputum; it became ill and was killed seventeen days later. *Post Mortem:* Tuberculosis of lymphatic glands, spleen and liver. From the spleen a salt suspension was made, and of this 2 c.cm. were injected into the base of the wing of two pigeons (11, 12). One bird was killed after ten days. *Post Mortem:* A small necrotic patch was found in the pectoral muscle at the site of injection;

smears from this showed lymphocytes, but no tubercle bacilli; the viscera were healthy. The second bird was killed sixteen and a half weeks after inoculation. *Post Mortem:* Bird well nourished; no evidence of disease was found in the body.

Experiment F.—Two guinea-pigs were injected subcutaneously with 2 c.cm. of a saline emulsion of sputum. One animal died four weeks later and was found to have tuberculosis of lymphatic glands, spleen and liver. The other guinea-pig was killed four weeks, also, after the inoculation; it was found to have contracted equally advanced tuberculosis; tubercle bacilli were present in smears from the spleen. The spleen was made into a suspension with salt solution. With this two pigeons (13, 14) were injected, each receiving 2 c.cm. at the base of the wing. The birds were killed eleven and a half weeks later. *Post Mortem:* Both were in good condition; there was no local nodule and no evidence of disease in the body.

Summary of the Results of the foregoing Series of Experiments.

The guinea-pigs injected with tuberculous sputum showed a rapid tuberculosis in every case except one, where the animal died within a week.

The pigeons inoculated from the tubercular material of these guinea-pigs were killed within the following times, and with the results set forth:—

3 days	*Nil.* A suspension of the spleen of this bird injected into a guinea-pig produced advanced tuberculosis in six weeks.
8 days	Local abscess.
10 days	Local necrotic patch.
6 weeks	Intrapectoral injection. *Nil.*
6½ weeks	Local necrotic patch. From this necrotic substance another pigeon was injected; this bird was killed 6½ weeks later; it showed no disease upon post-mortem examination.
8½ weeks	Two birds. *Nil.*
9 weeks	Two birds. *Nil.*
10 weeks	Two birds. Injection made into pectoral muscle. *Nil.*
11½ weeks	Two birds. *Nil.*
16½ weeks	*Nil.*

It may be observed that these results bring out a retrogression instead of a progression of the infective process, the most pronounced local lesions being those of the earlier dates; the birds killed at 8½ weeks and onwards showed, in fact, no local or other disease.

The high resistance of birds to infection with the human bacillus is thus forcibly illustrated.

THE FEEDING OF PIGEONS WITH TUBERCULOUS MATERIAL FROM BIRDS.

The birds used in these experiments were kept quite apart from those fed or inoculated with human sputum. They were housed in cages in a new wooden shed fitted up on the roof of the school building of St. Thomas's Hospital, on which no animals or birds of any kind had before been kept. Six pigeons were fed with the tubercular livers and spleens of various birds, mostly from the Gardens of the Zoological Society. The general food was grain. The tuberculous material was stored, as it came to hand, in stoppered bottles, in the cold ; portions were cut off as required with scalpel and forceps (boiled on each occasion both before and after the feeding) and then divided into fragments of moderate size, in a glass dish sterilised by boiling. Into the mouth of each bird about half a dozen of the pieces were inserted with a long pair of sterilised forceps.

Before describing the results of these experiments we may give a list of the birds which furnished the feeding material, and note under each the number of days the feeding was carried out. In every case the presence of tubercle bacilli was ascertained before the material was used.

(1) *Cormorant* (*Phalacrocorax carbo*).—Spleen, liver, and in this case the lungs, showed advanced tubercular disease. No ulcers of the intestine. Smears and sections of the liver showed typical bacilli. The left lung, with the exception of the apex, was converted into a tough yellowish caseating mass ; the lower half of the right lung presented a similar condition. From this material the pigeons were fed on November 3, 5, 6, 7, and 9.

(2) *Indian Tree Duck* (*Dendrocygna javanica*).—Liver and spleen showed advanced tubercular disease ; lungs not affected. Microscopic sections of the liver showed large numbers of tubercle bacilli. Birds fed November 10 and 12.

(3) *Domestic Fowl* (*Gallus bankiva*).—Spleen and liver strewn with tubercles of comparatively small size ; lungs healthy. On the outer aspect of the intestines were four or five nodules about as large as Barcelona nuts ; these were ulcerated on the side towards the lumen ; there were in addition three or four similar nodules of the size of a split pea, and one or two minute ulcers, each on a raised area representing an infected solitary gland. Microscopic examination of one of the larger excavated intestinal nodules showed the lesion to be full of tubercle bacilli. Birds fed November 13, 14, and 15.

(4) *Golden Eagle* (*Aquila chrysaëtus*).—Liver and spleen diseased. Microscopic examination of both showed large numbers of tubercle bacilli. Birds fed November 16, 17, 19, 20 and 21.

(5) *Common Pheasant* (*Phasianus colchicus*).—Liver and spleen enlarged, and in an advanced stage of disease. Lungs unaffected. Intestines, typical tubercular ulcers of mucosa. Microscopic examination of the spleen showed tubercle bacilli. Birds fed November 22, 23, and 24.

(6, 7) *Common Pheasant* (two birds).—In each, the spleen and liver were enlarged and full of yellow tubercles. Lungs unaffected. Intestine, circular ulcers of mucosa with raised edges. A smear of the spleen showed tubercle bacilli. Birds fed November 28, 29, and 30, December 1, 3, and 4.

(8) *Indian Porphyrio* (*Porphyrio calvus*).—Liver and spleen contained many caseating nodules; mesenteric glands enlarged and caseous. Intestines, no macroscopic disease. Smears from the spleen showed tubercle bacilli. Lungs unaffected. In the muscles outside the thorax there was a firm whitish mass, smears from which also showed acid-fast bacilli. Birds fed December 14.

(9) *Common Pheasant*.—Marked tuberculosis of the spleen and liver; microscopic sections of the former showed tubercle bacilli. Lungs unaffected. Intestine: In the mucosa there were a few small ulcers. There was a caseous plaque on one side of the thorax in front of, but not involving the lung. Birds fed January 3, 4, 5, and 7.

(10) *Phasianus scintillans*.—Well-marked tuberculosis of the liver and spleen. Bacilli in the lesions. Lungs unaffected. No macroscopic disease of the intestine. Birds fed January 24, 25, 26, and 28.

(11) *Domestic Fowl*.—Spleen and liver markedly diseased. Tubercle bacilli abundant in smears. Birds fed February 8, 9, 11, 12, 13, and 15.

(12) *Golden Pheasant* (*Thaumalea picta*).—Liver, which was full of small yellow tubercles, used. Smears show acid-fast bacilli. Birds fed March 2, 4, and 5.

(13) *Touraco* (*Turacus ?*).—Liver and spleen diseased; both contained tubercle bacilli. Birds fed April 17.

(14) *Mantchurian Crossoptilon* (*Crossoptilum mantchuricum*).—Liver and spleen full of tubercles. Smears of the liver showed an abundance of tubercle bacilli. The intestine showed a subpolypoid condition of mucosa, each elevation being due to the presence of a minute, though macroscopic, round worm. Birds fed May 2, 3, 4, 6, 7.

(15) *Tree Duck.*—Liver and spleen much diseased. Smears from the liver showed an abundance of acid-fast bacilli. Lungs unaffected. Birds fed May 25, 27, 28, 29.

(16) *Common Rhea* (*Rhea americana*).—Liver and spleen extensively diseased. Smears showed an abundance of tubercle bacilli. No peritoneal infection; some diffuse inflammation of small intestine; large intestine dark and congested; about six inches from rectum two small eminences project from the mucosa; these contained large numbers of tubercle bacilli. Bird fed June 7, 8.

(17) *Gannet* (*Sula bassana*).—Liver full of small yellowish tubercles; smears showed acid-fast bacilli. Bird fed June 19, 20.

Examination of the six Pigeons fed with Avian Tubercle.

Pigeon 1.—Killed December 31, 1906, after six weeks. All the viscera quite healthy. The intestine was slit up from end to end; no enlargement of the solitary glands, or other lesion, was present. No microscopic examination of the intestine was made.

Pigeon 2.—Killed January 26, 1907, after twelve weeks. Bird well nourished. No disease of liver, spleen, or lungs. Mucosa of small intestine for a distance of over 12 inches bright red; beyond this there is nothing abnormal. There are no ulcers in the congested area, but it is mottled with paler points, in places confluent so as to produce slightly larger areas; under a pocket lens these points appear greyish, somewhat translucent, slightly projecting, and suggestive of enlarged solitary glands. There are no tubercles in the peritoneal coat. In the congested part of the intestine there were about half a dozen round worms, each about 2 inches in length; these were identified by Dr. Louis Sambon as *Heterakis maculosa*. Microscopic examination of the intestine: in one set of sections the mucosa shows a series of spheroidal foci of lymphatic tissue in which groups of endothelial and giant-cells occur.[1] The foci in question project in varying degrees from the free surface, and, judging from their somewhat regular disposition and other characters, must be viewed as infected solitary lymph follicles. There is no involvement of the muscularis or of the peritoneum. Tubercle bacilli are present in large numbers in the endothelial and giant-cells; others lie free in the lymphatic tissue of the diseased follicles. Other sections display a notable enlargement of certain of the villi from tubercular infection; in the groups of endothelial cells in such infected villi clusters of acid-fast bacilli are present. This holds true invariably of the giant-cells occurring in the various lesions found in the pigeon after infection with avian tubercle, whether brought about by feeding or by the injection of salt suspensions of avian material; and it is true also of the spontaneous disease in the bird.

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[1] The nuclei of the giant-cells are usually confined to the more central part of the cell, which presents no necrotic centre.

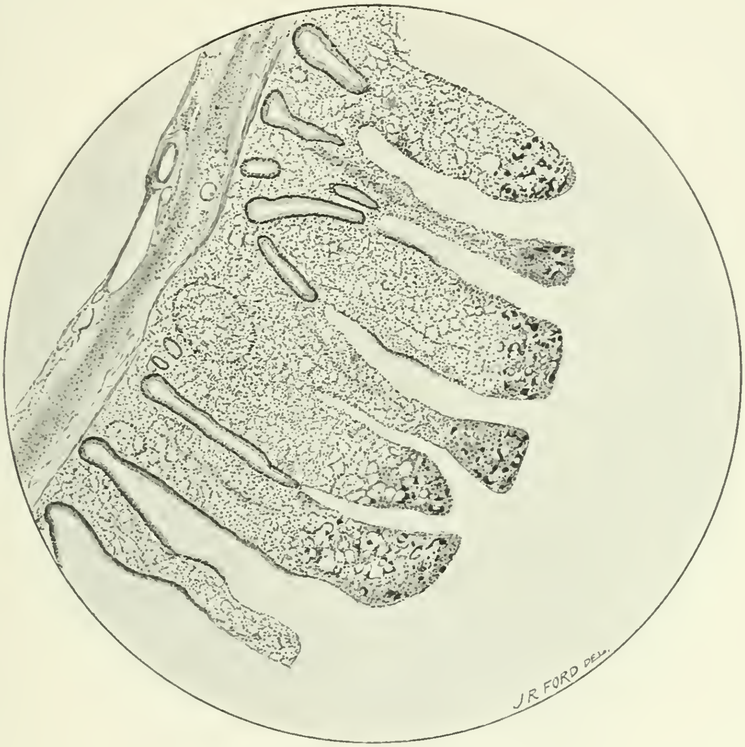

FIG. 4.

A microscopic section of portion of the intestine of a pigeon (No. 2) which had been fed for twelve weeks, at intervals, with the tubercular spleens and livers of various birds. Certain of the villi are considerably enlarged and polypoid from tubercular infection. The black foci represent the dense clusters of acid-fast bacilli in the endothelial cells of the affected villi. Stain: hot carbol fuchsin, followed by 25 per cent. nitric acid; counter-stain, hæmalum.

The technique adopted for the demonstration of the bacilli was invariably the following: the sections (cut in paraffin) were stained with hot carbol fuchsin, three changes, two minutes each; washed in water, treated with 25 per cent. nitric acid, washed in water, and counter-stained with hæmalum.

Pigeon 3.—Killed March 2, 1907, after seventeen weeks. Nothing abnormal was discernible in the solid viscera or intestine to the naked eye. The spleen was ground with sterilised salt solution, and 1 c.cm. of the suspension was injected into the subcutaneous tissue of the thigh of each of two guinea-pigs. One of these animals died fourteen days after the injection: no lesions were found upon examination. The second was killed nine weeks after the injection: no local lesion was discovered, and no disease of the abdominal or thoracic viscera.

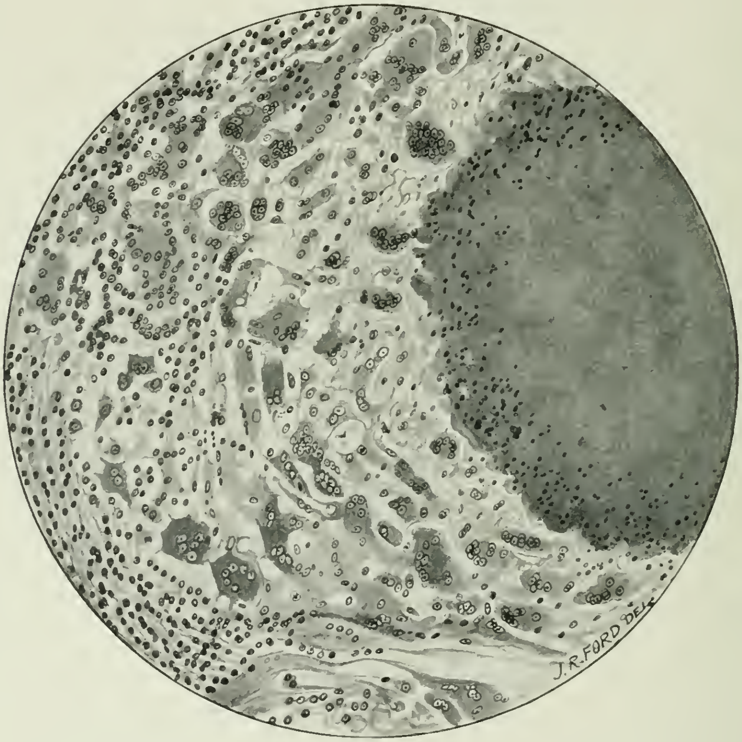

FIG. 5.

A microscopic section of portion of a tubercle in the deeper part of the intestinal mucosa of a pigeon (No. 3) which had been fed for seventeen weeks, at intervals, with the tubercular spleens and livers of various birds. Immediately around the caseous, necrotic centre there is a zone strewn with the chromatin of fragmented nuclei; beyond this lies the living portion of the tubercle. The number of giant-cells composing the last mentioned is very striking; their nuclei, however, show no circumferential disposition as they do in human tubercular giant-cells.

Microscopic examination of the intestine of this pigeon reveals the presence of tubercular disease of the solitary follicles and rows of well-defined tubercles confined to the peritoneal wall. In a few instances the peritoneal tubercle extends through the muscularis to a solitary follicle of the mucosa, the disease having reached the peritoneum *ex contiguo*. The larger and older of the peritoneal tubercles have a caseous centre; around this there is a zone strewn with fragments of chromatin, and beyond this the mass of endothelial cells, amongst which giant-cells occur in remarkable numbers. A few of the villi are enlarged and polypoid from tubercular infection.

The bacillary stain reveals the presence of large clusters of acid-fast bacilli in the endothelial and giant-cells as well as in the necrotic substance, in which the distribution of the clusters shows that the bacilli still retain an intracellular position. Bacilli occur also in large numbers in the groups of endothelial cells of those villi which are enlarged from disease. The number of tubercle bacilli in the endothelial and giant-cells in these preparations is very striking, and the same remark applies generally to the intestinal lesions of the pigeons infected with avian material. In actual numbers they can only be compared with the bacilli found in the cells of leprosy.

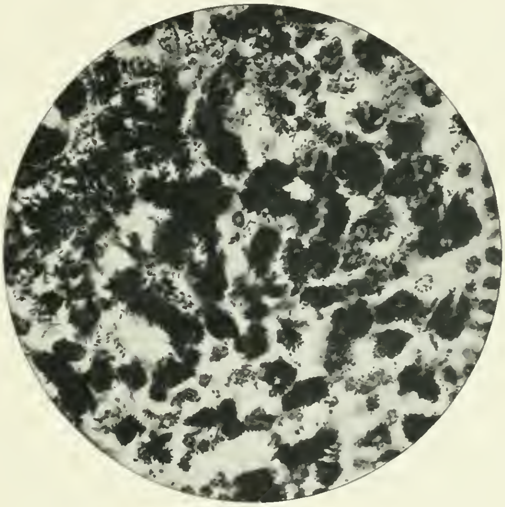

FIG. 6.

A microscopic section of an avian lesion showing the dense clusters of bacilli which are so regular a feature in avian tuberculosis. $\frac{1}{6}$ objective.

Pigeon 4.—Killed March 22, 1907, after twenty weeks.

Post Mortem.—Bird very thin. Lungs healthy; spleen, liver, present no macroscopic lesions. Microscopic examination of the spleen, however, reveals circular foci of endothelial cells, amongst which giant-cells occur; in these foci there are large numbers of tubercle bacilli. Intestine: the peritoneal coat shows numerous discrete tubercles scattered over its whole length, though most abundantly in the upper part, and commencing immediately beyond the gizzard. About 8 inches below the gizzard there were five round worms in the bowel, of the kind found in pigeon No. 2. Microscopic examination of the intestine reveals very extensive tubercular disease of the mucosa, which has in places extended by continuity through the muscularis to the peritoneal coat. Many of the villi are enlarged from similar disease. The several lesions are densely filled with acid-fast bacilli.

Pigeon 5.—Killed April 20, 1907, after twenty-four weeks. Bird wasted and ill. In this bird, in addition to the disease of the intestine, the spleen, liver and lungs presented microscopic lesions.

Post Mortem.—Spleen: not enlarged, but near one pole there was evident an opaque deeply-seated tubercle. Liver: studded on the surface and in section with minute opaque points which microscopic examination shows to be tubercular lesions containing acid-fast bacilli. Kidneys and ovary normal. Lungs: on the surface of each are three or four firm grey "tubercles," and a very few similar ones are scattered through the pulmonary substance; no pulmonary consolidation. Microscopic examination: although these lesions presented the histological appearances of tuberculosis with caseous centre, the bacillary stain did not demonstrate the presence of acid-fast bacilli. No tuberculosis of parietal peritoneum. Intestine: over the peritoneal surface there are scattered miliary tubercles; these become very sparse towards the anal end. The tubercles appear as distinct hemispherical or hemilenticular elevations on the peritoneal surface; as seen on section they do not involve the mucosa. In the mucosa there are numerous punctiform hæmorrhages or points of congestion. The intestine is not congested as a whole on either aspect. No projecting glands appear on the inner surface, and no ulcers. About 3 inches beyond the loop including the pancreas the intestine contained three round worms of the usual kind. Microscopic examination of the intestine reveals the presence of extensive disease of the mucosa. The disease extends through the muscularis (which is in places so infiltrated as to be no longer recognisable) to the peritoneum. The disease is in parts so extensive that its local origin is lost. Elsewhere lenticular tubercles occur in the peritoneum which are not continuous with any lesions in the mucosa. Many of the villi have undergone a polypoid enlargement from similar disease, and are infiltrated with endothelial and giant-cells. The several lesions are full of acid-fast bacilli.

Pigeon 6.—Killed June 21, 1907, after having been fed with avian tubercle at intervals for thirty-three weeks. Spleen not enlarged; at one pole there was a white focus as large as a hemp seed; the other solid abdominal viscera presented no lesions, neither did the lungs on surface or in section. Intestine: the peritoneal coat is thickly strewn with prominent opaque tubercles. These are most numerous in the upper part, immediately beyond the gizzard. For the terminal 4½ inches the small intestine is unaffected. Immediately beyond the cæca (both of which are enlarged and nodular from tubercular disease) the outer surface of the large intestine, which is about 1½ inches in length, is again thickly beset with tubercles. On one side of the cloaca there is a spherical mass, about 1 cm. in diameter, of firm, necrotic greyish-yellow substance. Microscopic examination of the intestine shows the presence of spheroidal tubercular foci in the mucosa and of peritoneal tubercles, the centres of some of which are caseous. In some instances the peritoneal tubercles are continuous with those of the mucosa, the intervening muscularis being destroyed. Large numbers of acid-fast bacilli are present in the several lesions. The lesions of the spleen and that by the side of the cloaca showed acid-fast bacilli and the usual histological evidence of tuberculosis.

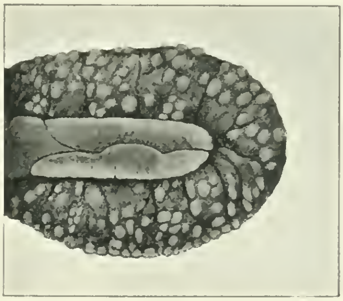

FIG. 7.

Portion of the loop of intestine, immediately beyond the gizzard, of a pigeon (*Columba livia*) which was fed, at intervals, for thirty-three weeks with the tubercular livers and spleens of seventeen birds of various kinds. The peritoneum is thickly covered with tubercles. (Mus. Royal College of Surgeons. Natural size.)

Summary of the Results of the preceding Group of Experiments.

Six pigeons were fed with avian tubercle at intervals, the pigeon kept the longest having received tubercular material from seventeen birds of various kinds.

Pigeon	Interval	Result
1	8 weeks	No macroscopic disease in any of the viscera.
2	12 weeks	No macroscopic disease in any of the solid viscera. Intestinal mucosa congested for a length of about 12 inches and studded with minute greyish elevations. Microscopical examination showed extensive tubercular disease of the mucosa, the lesions containing great numbers of acid-fast bacilli.
3	17 weeks	No macroscopic disease of solid viscera. Microscopical examination of the intestine showed extensive tuberculosis of the intestinal walls.
4	20 weeks	The peritoneal surface of the intestine thickly covered with small tubercles. Microscopical examination showed extensive tuberculosis of the different coats. Microscopical examination of the spleen revealed the presence of foci of endothelial cells and giant-cells, containing acid-fast bacilli.
5	24 weeks	Extensive tubercular disease of the peritoneum over the intestine. Microscopical examination showed extensive disease of the intestinal coats, the lesions being loaded with acid-fast bacilli. On the surface and through the liver were scattered a considerable number of minute opaque tubercles, which microscopical examination showed to contain acid-fast bacilli.
6	33 weeks	Very extensive tuberculosis of the intestinal peritoneum and other coats ; a tubercular focus in the spleen, and one by the side of the cloaca. Microscopical examination showed the presence of acid-fast bacilli and the usual histological characters of avian tuberculosis.

With the exception of the first bird, which was killed after eight weeks, but in which no microscopic examination was made of the intestine, intestinal infection took place in all. Disease of the spleen was observed after twenty weeks, and of the liver after twenty-four weeks.

The Transference of Charcoal Through the Intact Intestinal Mucosa of the Rabbit.

The transference of particulate material through the epithelium of an intact mucosa is a subject of high interest in connection with infective processes, and more particularly with tuberculosis. It has long been known that in the mesenteric tuberculosis of children, although the glandular disease may be far advanced, no lesion may be recognisable in the intestinal mucosa, notwithstanding that the latter must have been the entrance site of infection. The correctness of this assumption is confirmed by the experiments of Sidney Martin, published in the *Report of the Royal Commission on Tuberculosis*, 1895, Part III., Appendix, pp. 18 and 19. Martin proved that in pigs and calves tuberculosis of the lymphatic structures in the intestinal wall and elsewhere might be brought about by the ingestion of tuberculous material or phthisical sputum, without surface lesion.

In the *Transactions of the Pathological Society*, vol. liii., 1902, one of us has recorded, also, a case of œsophageal tuberculosis in a python, in which, without any ulceration, the "solitary glands," which in the ophidian œsophagus lie immediately beneath a simple columnar epithelium, were greatly enlarged from direct infection, the epithelium allowing of a ready migration of leucocytes between its cells. Although in our experiments, in which pigeons have contracted tuberculosis after the ingestion of avian tubercle, there has been no ulceration of the mucosa, the lymphatic structures of the mucosa have, nevertheless, been extensively infected, and in some cases, in addition, the muscularis and peritoneum; in one the infection had reached the liver and in two the spleen. The common pigmentation of the bronchial glands in healthy persons is an apt example of the same phenomenon, viz., the transport of particulate material through an intact surface. In order to see whether a similar passage of inorganic material could be induced through the intestinal mucosa, we devised the method of administering carbon to young rabbits for prolonged periods. We may confine ourselves to the most marked result amongst a group of such experiments. The feeding of a very young rabbit was begun, September 13, 1905, the food comprising oats, cabbage, and a liberal supply of the finest levigated wood charcoal made into biscuit with flour and lard. The droppings soon acquired a black colour. The animal was killed after forty-six weeks, having consumed altogether about six pounds of charcoal.

Post Mortem.—Upon inspection of the intestine from the exterior, the Peyer's patches were found to present a grey or slaty mottling; this was still more obvious on slitting up the bowel and viewing it, after washing, by transmitted light. The glands at the root of the mesentery, some of the Peyer's patches, and the spleen were fixed for microscopic examination, as were also portions of the bone-marrow from the lower end of the shaft of the femur and the upper end of the shaft of the humerus, close to the articular enlargement, the marrow being here of a deep red colour. Many sections of the spleen and bone-marrow were examined in the expectation of finding transferred particles of carbon, but none were seen, although the sections of the marrow showed thick strands of medullary tissue between the fat-cells. The extraneous material, however, was discovered in the Peyer's patches and in the mesenteric glands, the intestinal mucosa itself being perfectly normal. We had thus succeeded in reproducing experimentally in the intestinal tract the common pigmentation occurring in the bronchial glands of healthy persons from the inhalation of carbon. To describe the appearances in detail :—

(1) Lymphatic gland at the root of the mesentery. Lying in the proper lymphocytic tissue there occur groups of endothelial cells holding a varying number of carbon particles, some of which are angular and of considerable size. Many, if not all, of these cells contain in addition, numerous spherical granules. These granules, seeing that they present a pale yellow colour in sections stained simply with hæmalum, may be regarded as pigmentary; the occurrence of pigment in the endothelial cells which bridge across the lymph sinus is a recognised fact. Apart from such cells which hold carbon particles, considerable numbers of similar large, granular endothelial cells lie amongst the looser cells of the lymph sinus or applied to the gland trabeculæ. The groups, or

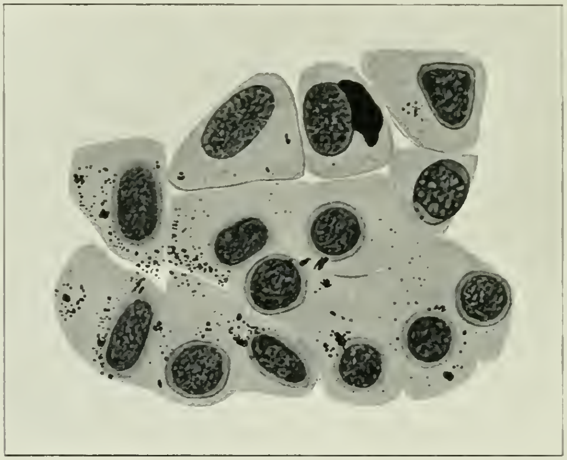

Fig. 8.

A nest of endothelial cells which lay in the lymphoid tissue of one of the glands from the root of the mesentery of a rabbit which was fed for forty-six weeks with charcoal biscuit. The cells hold particles of vegetable charcoal which have been transferred from the intestine. Hæmalum and eosin. $\frac{1}{12}$ oil immersion.

nests as they might be called, of carbon-holding cells do not lie in the lymph sinus, though at no great distance from it, but in the proper, close, lymphatic tissue; they are of comparatively small dimensions; they are not germ centres.

It is not to be imagined that the foreign particles have been actually translated in these cells from the intestine to the gland. They have, it must be assumed, either been discharged by carrier cells in the Peyer's patches and conveyed in a free state along the lymph stream to the glands, within which they have again been ingested by the endothelial cells, or they have been transferred to the gland in leucocytes which have therein been themselves ingested by the endothelial cells.

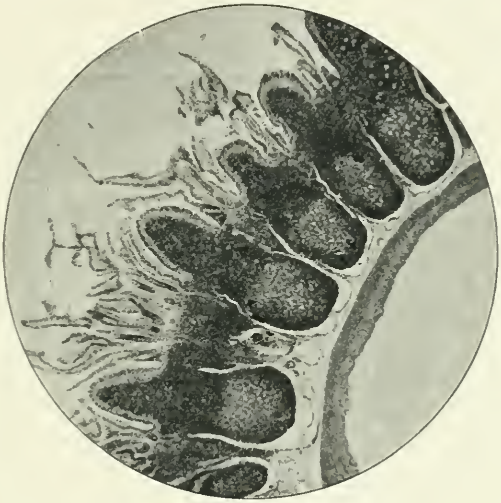

FIG. 9.

Portion of one of the Peyer's patches of the intestine of a rabbit which was fed for forty-six weeks with charcoal biscuit. In the deeper parts of several of the follicles composing the patch are dense foci of carbon particles. The mucosal epithelium is everywhere intact. 1 inch objective.

(2) Peyer's patch. In the deeper parts of many of the follicles composing the patch there are foci of carbon particles, the foci being of a size just visible to the naked eye. In the midst of the lymphatic tissue of the follicles, moreover, fine particles of the same substance, some in dense numbers, others in sparse, occur in isolated cells. The particles lie in sharply defined spaces, which may be single or multiple, and which present a dull yellowish colour, although no structure can be discerned in them. A careful study shows that the spaces are mostly, if not all, in large endothelial cells, for in some cases the displaced oval

nucleus and periphery of the polyhedral body of the cell can be distinctly seen. That the vacuole, however, is not strictly empty appears not only from its yellowish coloration, but also from the fact that a narrow cleft at times intervenes between the substance within it and the proper wall of the containing cavity. As in the case of the mesenteric glands, so here, the carbon has doubtless been taken up from the lumen of the intestine by leucocytes, which have wandered between the epithelial cells and thence back into the lymphatic tissue, in which they have been ingested by the endothelial phagocytes. That this is so appears from the nature of the material in the vacuoles.

Ruffer[1] has described very fully such a process of double intussusception as taking place in the dog's tonsil in connection with foreign particles at its surface, and has followed the different stages in the destruction of the leucocytes ingested by the deeper endothelial cells or macrophages. He described the nuclei of the ingested cells as finally disappearing, and the leucocytes as being then represented by yellowish-grey or dirty-brown spherical masses, which may attain a considerable size from swelling up of the cell. Ruffer figures such spheres singly and in groups in the macrophages; bacilli may or may not be recognisable in the digested remains of the leucocytes.

Some of the carbon particles lie free in the lymphatic tissue of the Peyer's patch. The most likely explanation of this is that after their conveyance from the surface by leucocytes they have been ejected by these, or by endothelial phagocytes into which they have been secondarily received; in this freed state the particles might be again ingested *in situ* or carried to the neighbouring lymphatic glands.

We have found carbon-holding cells in the follicles composing the Peyer's patch in a rabbit which was killed fifteen weeks after the same mode of feeding as that adopted for the preceding, the charcoal having been given from the time the animal was quite young.

THE INOCULATION OF PIGEONS WITH SALT SUSPENSIONS OF TUBERCULAR ORGANS FROM OTHER BIRDS.

Pigeon 1.—Injected partly subcutaneously and partly into the muscle with 2 c.cm. of a salt suspension of the tuberculous liver of a tree duck.[2] Killed four and a half weeks later.

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[1] *Quarterly Journal of Microscopical Science*, 1890, xxx., p. 481.

[2] In all cases the presence of acid-fast bacilli was demonstrated in the material before it was used as an injection. The site of injection was the base of the wing, the injection being partly subcutaneous, partly intramuscular.

P.M.—Caseous patches in muscle at the site of injection, microscopic sections of which showed the histological lesions of tuberculosis, and acid-fast bacilli. Liver and spleen unaffected.

Pigeon 2.—Injected with 2 c.cm. of a salt suspension of the tubercular liver of a cormorant. Killed six weeks later.

P.M.—No local disease ; no evidence of disease elsewhere.

Pigeon 3.—Injected with 2 c.cm. of a salt suspension of the tubercular spleen of the same cormorant. Killed six weeks later.

P.M.—No local or other disease.

Pigeon 4.—Injected with 2 c.cm. of a salt suspension of the tubercular liver of a tree duck. Killed after seven weeks.

P.M.—A local tuberculosis of muscle at site of injection, more advanced than in pigeon No. 1 injected from the same material and killed four-and-a-half weeks afterwards. Spleen and liver not diseased.

Pigeon 5.—Injected with 2 c.cm. of a salt suspension of the tubercular spleen of a pheasant. Killed nine weeks later.

P.M.—An extensive tubercular lesion at the site of injection, smears and sections of the muscle from which showed tubercle bacilli. Spleen and liver not diseased.

Pigeon 6.—Injected with 2 c.cm. of a salt suspension of the tubercular liver of a cormorant. Killed after eleven weeks.

P.M.—No local lesion, or lesion elsewhere.

Pigeon 7.—Injected with 2 c.cm. of a salt suspension of the tubercular spleen of a pheasant. Killed thirteen and a half weeks later.

P.M.—An extensive patch of caseation in the pectoral muscle. Spleen slightly enlarged, and containing several small tubercles. Smears from the local muscular lesion and from the spleen showed the presence of tubercle bacilli. Liver not diseased.

Pigeon 8.—Injected with 2 c.cm. of a salt suspension of the tubercular spleen of a pheasant. Killed fourteen weeks later.

P.M.—Extensive local lesion in muscle; liver showed several small foci, microscopic sections of which revealed the presence of tubercle bacilli. The spleen showed no microscopic disease.

Pigeon 9.—Injected with 2 c.cm. of a salt suspension of the tubercular spleen of a cormorant. Killed fifteen weeks later.

P.M.—Several small areas of caseation, scattered in the pectoral muscle about the site of injection. Microscopical sections of these showed the histological marks of tuberculosis and the presence of tubercle bacilli. Microscopical sections of the liver and spleen revealed no evidence of infection.

Pigeon 10.—Injected with 2 c.cm. of a salt suspension of the tubercular spleen of a pheasant. Killed after sixteen weeks.

P.M.—An extensive local lesion in the muscle; spleen enlarged and containing numerous tubercles, which showed acid-fast bacilli.

Pigeon 11.—Injected with 2 c.cm. of a salt suspension of the tubercular muscle of a pigeon which had received an injection of a salt suspension of the tuberculous spleen of a pheasant. Killed after twenty-one and a half weeks.

P.M.—Extensive local lesion in muscle; spleen full of tubercles; liver contains many small, discrete tubercles. Smears from the local abscess show masses of tubercle bacilli. Acid-fast bacilli were present also in the hepatic lesions.

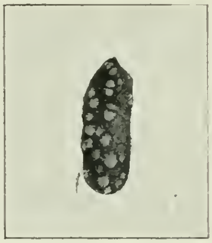

FIG. 10.

The spleen of a pigeon (*Columba livia*) in which this organ became markedly diseased after the subcutaneous and intramuscular injection of a salt suspension of avian tuberculous material. (Mus. Royal College of Surgeons. Natural size.)

Pigeon 12.—Injected with 2 c.cm. of salt suspension of the same tubercular muscle as that used in the foregoing experiment. Killed after twenty-one and a half weeks.

P.M.—Extensive local lesion in muscle. Spleen full of tubercles; liver shows nothing abnormal to the naked eye. Smears from the local lesion show masses of tubercle bacilli; sections of the liver show none.

Pigeon 13.—Injected with salt suspension made from the tubercular viscera of a pheasant. Killed after twenty-three weeks.

P.M.—Local lesion; tuberculosis of spleen, microscopical examination showing tubercle bacilli in the lesions.

(In this experiment the injection was made strictly into the subcutaneous tissue, the muscle not being injured.)

Pigeon 14.—Injected with the same salt suspension as that used in the preceding experiment. Killed after thirty and a half weeks.

P.M.—Local tuberculous ulcer; spleen showed numerous tubercles scattered through it. Smears from the ulcer showed masses of acid-fast bacilli. Microscopical examination of the liver disclosed no tubercular lesions.

(In this experiment the injection was made strictly into the subcutaneous tissue, the muscle not being injured.)

It may be observed that the tubercular material furnished by the different birds used did not prove equally virulent. The least virulent was that from the cormorant, and in this bird the number of bacilli present in smears made from the lesions was particularly small.

Summary of the Results of the foregoing Group of Experiments.

Pigeon	Period	Result
1	4½ weeks	Local lesion.
2	6 weeks	*Nil.*
3	6 weeks	*Nil.*
4	7 weeks	Extensive local lesion.
5	9 weeks	Extensive local lesion.
6	11 weeks	*Nil.*
7	13½ weeks	Local lesion and disease of spleen.
8	14 weeks	Local lesion and disease of liver.
9	15 weeks	Local lesion.
10	16 weeks	Local lesion and disease of spleen.
11	21½ weeks	Local lesion and disease of spleen and liver.
12	21½ weeks	Local lesion and disease of spleen.
13	23 weeks	Local lesion and disease of spleen.
14	30½ weeks	Local lesion and disease of spleen.

The inoculation of pigeons with salt suspension of the tubercular organs of birds produces a local lesion, followed, after thirteen weeks, by disease of the spleen, and, it may be, of the liver. In a further pigeon, a pure culture of avian bacillus was used as the injection, in place of tuberculous organs.

Pigeon 1.—A suspension of the avian bacillus was injected intraperitoneally.[1] Killed after fourteen and a half weeks.

P.M.—Extensive peritoneal tuberculosis, accompanied with matting of the viscera; the tubercles were scattered on the surface of the spleen, liver, and intestine, and in the substance of the first-named organ. Smears from the divided surfaces of the lumbar glands showed masses of tubercle bacilli lying in numerous large cells. The right lung was also infected.

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[1] The cultures used were some carried on from one kindly supplied to us by Professor Delépine and raised by him from a domestic fowl.

THE INOCULATION OF GUINEA-PIGS WITH SUSPENSIONS OF TUBERCULOUS MATERIAL FROM BIRDS.

Guinea-pig 1.—Of a salt suspension of the tuberculous spleen of a pheasant, 2 c.cm. were injected into the leg of a guinea-pig. The animal died nine days later.

P.M.—No lesion at the site of inoculation or elsewhere in the body.

Guinea-pig 2.—Injected subcutaneously with 2 c.cm. of a salt suspension of the tuberculous liver of a tree duck. The animal was killed ten days later.

P.M.—Local tuberculous abscess; popliteal glands and inguinal enlarged. Smears from the caseous glands showed tubercle bacilli. Liver and spleen normal.

Guinea-pig 3.—Injected into leg with a salt suspension of the tuberculous liver of the Egyptian goose. The animal was killed after ten days.

P.M.—Abscess at seat of inoculation; the inguinal and lumbar glands affected, microscopic examination showing the presence of cells full of acid-fast bacilli.

Guinea-pig 4.—Injected into leg with 2 c.cm. of a salt suspension of the tuberculous spleen of a pheasant. The animal died four and a half weeks later.

P.M.—No local abscess; popliteal gland enlarged, but not caseous. Viscera normal.

Guinea-pig 5.—Injected subcutaneously with 2 c.cm. of a salt suspension of the tuberculous muscle of a pigeon, into which 2 c.cm. of a salt suspension of the tuberculous spleen of a pheasant had been injected. The animal died four and a half weeks later.

P.M.—Local caseous abscess; tubercular infection of popliteal, inguinal and lumbar glands. Spleen and liver, no disease.

Guinea-pig 6.—Injected into leg with salt suspension of the tuberculous liver of an Egyptian goose. The animal was killed after five weeks.

P.M.—Local lesion and disease of the neighbouring lymphatic glands.

Guinea-pig 7.—Injected subcutaneously with 2 c.cm. of a salt suspension of the tuberculous liver of a cormorant. The animal was killed six weeks later.

P.M.—Abscess at site of injection; inguinal and lumbar glands enlarged, popliteal gland caseous; smears from the latter showed large numbers of tubercle bacilli.

Guinea-pig 8.—Injected subcutaneously with 2 c.cm. of a salt suspension of the tuberculous spleen of the same cormorant as used above. The animal was killed after six weeks.

P.M.—A minute caseous focus in muscle at site of inoculation; inguinal gland enlarged; smears of the gland showed the presence of tubercle bacilli. Liver and spleen normal.

Guinea-pig 9.—Injected subcutaneously with 2 c.cm. of a salt suspension of the tuberculous muscle of a pigeon into which 2 c.cm. of a salt suspension of the tuberculous spleen of a pheasant had been injected. The animal was killed after seven weeks.

P.M.—Tubercular disease of popliteal, inguinal and lumbar glands; smears from one of the caseous glands showed the presence of tubercle bacilli. Spleen and liver free of disease. A suspension of these glands was injected into two guinea-pigs; of these one died eleven weeks later. *P.M.*: a local abscess at site of inoculation, smears from which showed tubercle bacilli; no glandular enlargement; the other guinea-pig was killed twelve and a half weeks later. *P.M.*: A small necrotic patch at site of inoculation, which contained tubercle bacilli; viscera healthy.

Guinea-pig 10.—Injected into leg with 2 c.cm. of a salt suspension of the tuberculous spleen of a pheasant. The animal was killed after fourteen weeks.

P.M.—A local tubercular disease of inguinal glands; spleen and liver, nothing abnormal.

Guinea-pig 11.—Injected into leg with 2 c.cm. of a salt suspension of the tuberculous spleen of a pheasant. The animal was killed after twenty-two weeks.

P.M.—A considerable local swelling had developed subsequently to the inoculation, but this eventually completely disappeared; a minute necrotic patch was found in the muscle at the site of injection. There was no other evidence of disease in the body.

Summary of the Results of the foregoing Group of Experiments.

Guinea-pig	Interval	Result
1	9 days	No tuberculosis.
2	10 days	Local lesion and affection of neighbouring lymphatic glands.
3	10 days	Local lesion and affection of neighbouring lymphatic glands.
4	4½ weeks	No tuberculosis.
5	4½ weeks	Local lesion and disease of neighbouring lymphatic glands.
6	5 weeks	Local lesion and disease of neighbouring lymphatic glands.
7	6 weeks	Local lesion and disease of lymphatic glands.
8	6 weeks	Local lesion and disease of lymphatic glands.
9	7 weeks	Local lesion and disease of lymphatic glands.
10	14 weeks	Disease of the local glands.
11	22 weeks	Minute local lesion.

It appears from these results that the infective process, although it may produce a local lesion and a secondary involvement of the neighbouring lymphatic glands, does not extend to the abdominal or thoracic viscera, and this in spite of the fact that the time allowed in some of the experiments amounted to 22 weeks.

Another point worthy of remark is that, instead of an advance in the infective process, a retrogression appeared to take place when the animals were kept for considerable periods.

In both of these ways the results differ from those which follow the similar injection made upon the guinea-pig with human sputum.

As a corollary of these results which follow the inoculations of guinea-pigs with tuberculous material from birds, we may refer back to those attending the injection into pigeons of suspensions of the tubercular organs of guinea-pigs infected with human sputum, for under the second group the history of the infective process is also one of retrogression.

THE INOCULATION OF A GUINEA-PIG WITH A CULTURE OF THE AVIAN BACILLUS.

In the case of one further guinea-pig a pure culture of avian tubercle bacillus was used as the injection in place of a salt suspension of tuberculous organs.

Guinea-pig 1.—Injected intraperitoneally with a pure culture of avian tubercle bacillus.[1] Animal killed twelve and a half weeks later, at which date it was in perfect health.

Post Mortem.—Thickening of abdominal wall at site of injection; large numbers of tubercles in the great omentum, which was thickened and rolled up. Tubercles were present on the peritoneal surface of the spleen, liver, diaphragm, but none in the divided surfaces of the spleen or of the liver. No disease of the lymphatic glands or thoracic contents. Microscopic examination of the splenic tissue showed no evidence of tuberculosis and no tubercle bacilli. Sections of the omentum, and of one of the tubercles on the peritoneal surface of the spleen, showed the presence of large numbers of tubercle bacilli lying in rounded foci of acute inflammatory tissue, beyond which there was a zone of chronic inflammation, but none of the ordinary marks of a tubercular lesion.

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[1] The cultures used were some carried on from one kindly supplied to us by Prof. Delépine, and raised from a domestic fowl.

Summary of the Results of the foregoing Experiment.

One guinea-pig injected intraperitoneally with the avian bacillus showed, after twelve and a half weeks, a formation of tubercles containing bacilli on the omentum and on the peritoneum, without any deep involvement of viscera. It is by no means clear that a real infective process resulted in this case. The lesions did not resemble those proper to a true infective formation.

THE FEEDING OF A RHESUS MONKEY WITH AVIAN TUBERCULAR MATERIAL.

The animal was kept for a month after having been received, and, being to every appearance healthy: its feeding was commenced on January 3, 1907. Small portions of the tubercular spleens and livers of certain of the birds used for feeding the six pigeons already referred to were finely minced and inserted into the middle of slices of banana. Although the monkey was fastidious, he was induced to take a small amount of the tuberculous material so offered on each occasion. (1) Common pheasant: from the tubercular liver the animal was fed on January 3, 4, 5, 6, 1907; (2) *Phasianus scintillans*: fed January 25, 26, 28; (3) Domestic fowl: fed February 6, 8, 9, 12, 13, 15; (4) Golden pheasant: fed March 2, 4, 5; (5) Touraco: fed April 10, 12, 13, 15, 16, 17; (6) Double-spurred francolin: fed April 25, 26, 27; (7) Mantchurian crossoptilon: fed May 1, 2, 3; (8) Tree duck: fed May 25, 27; (9) Rhea: fed June 7, 8; (10) Gannet: fed June 19, 20. The animal remained in good health, and was killed with chloroform on July 8, 1907, twenty-six weeks after the commencement of the experiment.

Post Mortem.—No trace of disease was discovered in any of the abdominal or thoracic organs, and none in the intestine, which was slit up throughout and critically examined. There was no lesion of the peritoneum.

The Rhesus monkey is readily infected by means of human phthisical sputum. In Allan MacFadyen's experiments,[1] intestinal and more remote tubercular disease was regularly found after the administration of tuberculous sputum. The death of the animals occurred in different cases in thirty, forty-three, sixty-five days. In the last two the sputum was given only twice and once respectively. The monkey proved equally susceptible when fed with tubercular material from bovine source. The author points out that, whilst intestinal lesions were found in the case of every monkey that had received human sputum, none of the monkeys fed with the bovine material presented any evidence of tuberculous ulcers of the gut. This difference, we think, is possibly attributable to the circumstance that human tuberculous sputum contains other pathogenic micro-organisms than the tubercle bacillus, whilst the latter would be in a pure, or comparatively pure state, as given in the material from tubercular bovines. The possibility of infecting the Rhesus monkey by the administration of tuberculous cow's milk is shown by the results obtained in the work of the "Royal Commission on Tuberculosis (Human and Bovine)."[1] In one experiment, moreover, a single dose of not more than 1 mg. of a pure culture of the bovine bacillus was followed by infection.

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[1] *Lancet*, 1903, ii., p. 744.

Is the Human Tubercle Bacillus Convertible into the Avian?

The foregoing observations bring out unquestionably a marked difference in the pathogenicity of the human and the avian bacilli. The *human* bacillus is pathogenic to the bird to a very limited extent only; it is markedly pathogenic to the guinea-pig, and to the monkey. The *avian* bacillus is markedly pathogenic to the bird; it is scarcely pathogenic to the guinea-pig or to the monkey. Although these marked differences exist between the two bacilli, the question, nevertheless, arises how far the one is a form of the other modified by its insertion into, or passage through, another species of animal.

It is possible that the human bacillus, on its insertion into the pigeon, may lose certain of its characters, and become incapable of exciting more than a trivial disease in the guinea-pig, or in the human subject from whom it was originally derived. This would, of course, be nothing more than is witnessed in the results of human vaccination with calf lymph obtained by inoculating the calf with variolous pus. Were this so, it should follow that the human bacillus introduced into the pigeon would so lose its virulence that when transferred from the avian lesions, or from the intact avian organs, to the guinea-pig, it should set up in this animal, either no disease at all, or a modified form of disease differing from that which arises after the direct inoculation with human sputum, in being confined to the site of inoculation and the adjacent lymphatic glands. In the entire series of the experiments reported in the preceding part of this communication, there are only four that bear upon this theoretical possibility.

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[1] *Second Interim Report*, 1907, p. 12.

(1) A pigeon was inoculated at the base of the wing with a salt suspension made from the tubercular lymphatic glands of a guinea-pig, which was rendered tuberculous by the subcutaneous injection of 2 c.cm. of a thin salt emulsion of phthisical sputum, the animal dying in fourteen days, with a local caseous abscess and tuberculosis of the lymphatic glands. The pigeon was killed after three days.

Post Mortem.—Nothing was found at the site of inoculation, and no disease observed; the spleen, to the naked eye, was normal. Of this spleen a salt suspension was made and injected into the leg of a guinea-pig; the animal died after six weeks, with a local caseous abscess, and tuberculosis of the lymphatic glands, and of the spleen and liver. Smears from the abscess showed large numbers of tubercle bacilli; sections of the spleen showed typical lesions containing tubercle bacilli.

(2) A pigeon was fed almost daily for nineteen weeks with peas mixed with tubercular sputum. There were no macroscopic lesions of the viscera found after the bird was killed, except that the higher parts of the intestine showed patches of congestion, in which the solitary glands appeared as minute whitish elevations. There were no peritoneal tubercles. The spleen was made into a salt suspension and injected subcutaneously into the thigh of a guinea-pig. A nodule developed at the site of injection eleven days later; the inguinal glands enlarged. The animal died nine weeks after inoculation.

Post Mortem.—A small ulcer at the site of the inoculation; inguinal glands much enlarged; smears from one of the glands showed beaded acid-fast bacilli. Lumbar glands of the corresponding side enlarged. The spleen showed prominent groups of miliary tubercles, in which microscopic examination proved the presence of tubercle bacilli.

(3) A pigeon was injected at the base of the wing with a salt emulsion of tuberculous sputum. It developed a local lesion, and was killed after seven weeks. A salt suspension made from this lesion produced a limited tuberculosis in a guinea-pig in five weeks. A salt suspension from the latter lesion, on being carried to a second guinea-pig, produced marked tuberculosis and death in nine weeks.

(4) A guinea-pig was injected subcutaneously with 1 c.cm. of a salt suspension of the tuberculous muscle of a pigeon into which 2 c.cm. of a salt suspension of the tuberculous spleen of a pheasant had been injected. The animal was killed after seven weeks.

Post Mortem.—Tubercular disease of popliteal, inguinal and lumbar glands; smears from one of the caseous glands showed the presence of tubercle bacilli; spleen and liver free of disease.

A suspension of these glands was injected subcutaneously into two further guinea-pigs. Of these, one died eleven weeks later.

Post Mortem.—A local abscess at the site of the inoculation, smears from which showed tubercle bacilli ; no glandular involvement.

The other guinea-pig was killed twelve and a half weeks later.

Post Mortem.—A small necrotic patch at the site of the inoculation, which contained tubercle bacilli ; viscera healthy.

This fourth observation shows that the avian disease after inoculation into a pigeon produced in the guinea-pig a local and glandular disease as usual, and the further injection of the caseous material from this guinea-pig into two further guinea-pigs produced again only a local disease. The true avian bacillus was not transmuted so as to behave like the human to the guinea-pig after two transferences through the animal named.

We may next record, therefore, the following group of experiments which were devised in order to ascertain how far the characters of the human bacillus were modified by its passage through the bird ; whether after such a passage, *i.e.*, after its exposure to the cells and tissue juices of the bird, it would, if inoculated into the guinea-pig, reproduce the generalised disease which arises in that animal from the direct inoculation of human sputum ; or whether it would produce only a local and glandular disease, such as is set up in the guinea-pig by inoculation with the avian bacillus. In the case of each inoculated bird the spleen was used to inoculate the guinea-pig, and in the case of the later birds the local lesion produced at the site of inoculation was used, in addition, to inoculate a further series of guinea-pigs. Five pigeons received an injection into the base of the wing, of 1 c.cm. of a salt emulsion of phthisical sputum in which a plentiful supply of tubercle bacilli was demonstrated. The skin was first washed with ether ; the site of puncture was closed with collodion. The birds were killed at different periods, and the spleen in each case was rubbed up in a mortar with sterilised salt solution and injected into the guinea-pig. In the case of the last three of the five birds, the local lesion was, in addition, rubbed up with salt solution and used as an injection upon a further series of guinea-pigs.

Pigeon 1.—Killed July 30, 1907, seven days after inoculation with tuberculous sputum.

Post Mortem.—There was a local lesion at the site of inoculation, consisting of a dry flat plaque of yellow colour in the substance of the pectoral muscle. A smear made from this showed the presence of tubercle bacilli, though not in great numbers. The viscera, including the spleen, were unaffected, to the naked eye. The spleen was removed with sterilised scissors and forceps, and rubbed up in a mortar with sterilised salt solution.[1] The whole 3 c.cm. was injected into the muscles of the calf of a guinea-pig. The guinea-pig was killed September 30, fifty-two days after inoculation.

Post Mortem.—Local lesion; in the muscles of the calf there was a soft, cheesy, encapsulated abscess. Lymphatic glands: the popliteal and the superficial and deep inguinal glands were enlarged and caseous. Spleen: much enlarged; strewn, both on the surface and in section, with miliary tubercules. Liver: surface and section studded with small tubercles. Lungs: both studded on the exterior and in section with typical grey tubercles.

Pigeon 2.—Killed August 6, 1907, fourteen days after inoculation with tuberculous sputum.

Post Mortem.—There was a local lesion appearing as a small, dry, yellowish, necrotic mass, readily enucleated from the muscle in which it lay. Smears showed but very few tubercle bacilli. All the organs, including the spleen, were normal to the naked eye. The spleen was rubbed up with sterilised salt solution and injected (1 c.cm.) beneath the skin of the thigh of a guinea-pig. The guinea-pig was killed September 30, 1907, fifty-five days after its inoculation.

Post Mortem.—Local lesion; a small superficial ulcer. Lymphatic glands: the superficial and deep inguinal, and the lumbar glands of the same side, were enlarged, and caseous on section. Spleen: showed four typical prominent yellowish tubercles; the organ, as a whole, not enlarged. Liver: nothing visible on the surface; on careful slicing one miliary tubercle was found, which in microscopic section showed a small necrotic centre surrounded by endothelial cells and a few giant cells, between which were a certain number of polymorphonuclear leucocytes. Lungs: one typical grey tubercle at the surface, the diagnosis of which was confirmed microscopically.

Pigeon 3.—Killed August 20, 1907, four weeks after inoculation with tuberculous sputum.

Post Mortem.—The local lesion appeared as a foul cheesy abscess, with a capsule of connective tissue. Smears showed tubercle bacilli; the skin was intact, the abscess being intramuscular. The spleen was rubbed up with sterilised salt solution, and the whole (2 c.cm.) was injected into the right thigh of a guinea-pig. The spleen presented no disease to the naked eye.

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[1] In all these experiments the instruments, mortar, and other vessels were invariably boiled immediately before use, and the salt solution was boiled likewise on each occasion.

A second guinea-pig was injected, subcutaneously in the thigh, with a salt suspension of the caseous contents of the intramuscular abscess of the same pigeon. Both guinea-pigs were killed on October 15, 1907, fifty-six days after their inoculation, and both showed, on *post-mortem* examination, widespread tuberculosis.

(1) The guinea-pig injected subcutaneously with the spleen of the pigeon showed: local lesion; an intramuscular caseous focus at the site of injection. Lymphatic glands: superfical and deep inguinal, and lumbar, enlarged and caseous. Spleen; much enlarged, uniformly studded with yellow, prominent tubercles. Liver: shows many tubercles scattered at the surface and in section. Lungs: grey tubercles at the surface and in section.

(2) The guinea-pig injected with the local tuberculous lesion of the same pigeon. Local lesion; abscess at the site of inoculation. Lymphatic glands: superficial and deep inguinal, and lumbar glands, enlarged and caseous. Spleen: large and full of tubercles. Liver: a certain number of tubercles on the surface and in section. Lungs: miliary tubercles freely scattered at surface and in section.

Pigeon 4.—Killed September 3, 1907, six weeks after inoculation.

Post Mortem.—The local lesion consisted of a small mass of putty-like encapsulated material, smears from which showed the presence of tubercle bacilli. None of the viscera exhibited any signs of disease.

One guinea-pig was inoculated with a salt suspension of the local lesion; another was inoculated with a salt suspension of the bird's spleen, which presented no marks of disease. The injection was made into the back of the leg. The animals were killed October 29, 1907, fifty-six days after inoculation.

(1) That into which a salt suspension of the local lesion had been injected showed—Local: an intramuscular, firm, partly caseous focus in the muscles of the leg. Lymphatic glands: enlargement and caseation of superficial and deep inguinal glands, and of the lumbar of the same side. Spleen: slightly enlarged, surface granulated from the presence of small tubercles; at one pole the tubercles are larger and more prominent; small grey tubercles appear in section. Liver: about half-a-dozen opaque tubercles visible at the surface, and one or two in the sections. Lungs: no tubercles apparent on the surface or in section.

(2) The guinea-pig into which the apparently normal spleen of the bird was injected:

Post Mortem.—Local: abscess in muscles of calf; the popliteal, inguinal, and lumbar glands enlarged and caseous. Spleen: enlarged to three times the normal size, with prominent tubercles distributed throughout. Liver: two or three tubercles at the surface; none others seen in section. Lungs: surface strewn with grey tubercles; a few also in the sections.

Pigeon 5.—Killed September 17, 1907, eight weeks after inoculation.

Post Mortem.—The local disease appeared as a small elongated lesion. None of the viscera showed any signs of disease.

One guinea-pig was inoculated with the local lesion, rubbed up in sterilised salt solution; no microscopic smears were made from the lesion, owing to its smallness. Another guinea-pig was inoculated with a salt suspension of the bird's spleen, which presented no evidence of disease.

(1) The guinea-pig into which a salt suspension of the local lesion had been injected was killed on November 11, 1907, fifty-five days after inoculation.

Post Mortem.—Local: an intramuscular abscess, containing soft, putty-like material. Lymphatic glands: deep inguinal and corresponding lumbar, caseous; retroperitoneal and mediastinal, tubercular. Spleen: enlarged; surface granular from the presence of tubercles; section thickly strewn with miliary tubercles. Liver: surface and section closely strewn with miliary tubercles. Lungs: surface and section show abundant numbers of miliary tubercles.

(2) The animal into which the salt suspension of the same pigeon's spleen had been injected died on October 31, 1907, forty-four days after its inoculation.

Post Mortem.—Local: a caseous abscess in the muscles of the thigh. Lymphatic glands: moderate enlargement of the superficial and deep inguinal glands, which were caseous on section. Spleen: considerably enlarged, thickly studded with tubercles. Liver: Thickly strewn with tubercles. Lungs: a certain number of grey tubercles scattered over the surface.

Summary of the Results of the preceding Group of Experiments.

A series of guinea-pigs inoculated intramuscularly with the spleens of five pigeons which had received intramuscular injections of human tuberculous sputum contracted general tubercular disease, notwithstanding the fact that the spleens of the pigeons presented no macroscopic lesions. The disease set up in the guinea-pigs was as pronounced when the spleen was taken from the pigeon eight weeks after the pigeon's inoculation as when taken at the end of one week. In three cases the local lesion produced by sputum-injection in the pigeon was used, in addition to the spleen, to inject a further series of guinea-pigs; all the animals contracted general tuberculosis.

THE RELATION OF THE HUMAN TO THE AVIAN BACILLUS AS TESTED BY MEANS OF THE "OPSONIC" INDEX.

As the agglutination test, though originally introduced as a means of diagnosing the specific characters of a serum, may be conversely used to diagnose a bacillus, so may the opsonic phenomenon be used as a means of studying the characters of a serum, or conversely to study the characters of a bacillus. This method we have employed as a means of testing the human against the avian tubercle bacillus. As so applied, it consists in saturating samples of the serum of patients suffering from tuberculosis (i.) with a suspension of dead human bacilli; (ii.) with a suspension of dead avian bacilli; and afterwards estimating the amount of phagocytosis which occurs when to the saturated serum (deprived of its bacilli by centrifuging) there are added further bacilli of either kind, and the leucocytes of normal blood.

The question is, will the human bacillus which is used to saturate the tuberculous serum extract only the "opsonin" which acts upon the human bacillus, or will it extract likewise that which acts upon the avian? And, as a corollary, will the avian bacillus extract only the opsonin which acts upon the avian bacillus, or will it extract that also which acts upon the human?

In selecting the cases of human disease we took seven of pulmonary tubercle, as being representative of human tuberculosis; none of these had been treated by means of tuberculin. We took, in addition, two cases of tuberculous cystitis, treated with tuberculin; one of tuberculous meningitis, not treated with tuberculin; and two of tuberculous osteitis, treated with tuberculin.

We may first consider the amount of phagocytosis that takes place when the two bacilli are presented to the action of normal human leucocytes, in the serum of patients suffering from tuberculosis, after saturation of different samples of this serum with the human and avian bacillus respectively.

The human tubercle bacilli which were employed for these observations were either masses of dead bacilli, obtained direct from Dr. W. Bulloch, or living cultures of bacilli, which were killed by exposure to a temperature of 60° C. just before the experiments were commenced. The cultures of the avian tubercle bacilli were from a growth raised by Professor Delépine, and these were killed in a similar manner. In every instance, the suspensions of the bacilli were made in 1·5 per cent. sodium chloride, using tap water.

The human serum which was employed was in every instance quite fresh. It was usually obtained one or two hours before the experiments were commenced. The leucocytes used were those of normal human blood.

OBSERVATION I.—CASE OF PULMONARY TUBERCULOSIS IN WHICH NO TUBERCULIN HAD BEEN GIVEN.

A. Patient's serum + emulsion of human tubercle bacilli + human leucocytes:
50 cells contained 135 bacilli.

Equal portions of the serum and a *very thick* emulsion of human tubercle bacilli were digested for one and a half hours at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 20 bacilli.

(2) Equal portions of clear fluid + avian bacilli + human leucocytes:
50 cells contained 13 bacilli.

B. Patient's serum + emulsion of avian tubercle bacilli + human leucocytes:
50 cells contained 320 bacilli.

Equal portions of the serum and a *very thick* emulsion of avian tubercle bacilli were digested for one and a half hours at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 51 bacilli.

(2) Equal portions of clear fluid + avian bacilli + human leucocytes:
50 cells contained 34 bacilli.

OBSERVATION II.—CASE OF ADVANCED PULMONARY TUBERCULOSIS IN WHICH NO TUBERCULIN HAD BEEN GIVEN.

A. Patient's serum + emulsion of human tubercle bacilli + human leucocytes:
50 cells contained 150 bacilli.

Normal human serum + emulsion of human tubercle bacilli + human leucocytes:
50 cells contained 107 bacilli.

Opsonic index = 1·4.

Equal portions of the patient's serum and a *very thick* emulsion of human tubercle bacilli were digested for one and a half hours at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 15 bacilli.

(2) Equal portions of clear fluid + avian bacilli + human leucocytes:
50 cells contained 13 bacilli.

B. Patient's serum + emulsion of avian tubercle bacilli + human leucocytes:
50 cells contained 155 bacilli

Normal human serum + emulsion of avian tubercle bacilli + human leucocytes:
50 cells contained 74 bacilli

Opsonic index = 2·0.

Equal portions of the patient's serum and a *very thick* emulsion of avian bacilli were digested for one and a half hours at 37° C. and then centrifuged at high speed.

(1) Equal portions of clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 34 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 36 bacilli.

OBSERVATION III.—CASE OF ADVANCED PULMONARY TUBERCULOSIS NOT TREATED WITH TUBERCULIN.

A.—Patient's serum + emulsion of human tubercle bacilli + human leucocytes: 50 cells contained 151 bacilli.

Equal portions of the serum and a *very thick* emulsion of human tubercle bacilli were digested for two and a half hours at 37° C. and then centrifuged at high speed.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 32 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes: 50 cells contained 147 bacilli.

Equal portions of the serum and a *very thick* emulsion of avian tubercle bacilli were digested for two and a half hours at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 34 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 27 bacilli.

OBSERVATION IV.—CASE OF ADVANCED PULMONARY TUBERCULOSIS WHICH HAD NOT BEEN TREATED WITH TUBERCULIN.

A.—Patient's serum + emulsion of human tubercle bacilli + human leucocytes: 50 cells contained 175 bacilli.

Equal portions of the serum and an emulsion of human tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 66 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 39 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes: 50 cells contained 122 bacilli.

Equal portions of the serum and emulsion of avian tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 46 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 33 bacilli.

OBSERVATION V.—CASE OF ADVANCED PULMONARY TUBERCULOSIS.

A.—Patient's serum + emulsion of human tubercle bacilli + human leucocytes: 50 cells contained 160 bacilli.

Equal portions of the serum and an emulsion of human tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 43 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 23 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes:
50 cells contained 75 bacilli.

Equal portions of the serum and an emulsion of avian tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 53 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes:
50 cells contained 27 bacilli.

OBSERVATION VI.—CASE OF PULMONARY TUBERCULOSIS WHICH HAD NOT BEEN TREATED WITH TUBERCULIN.

A.—Patient's serum + emulsion of human tubercle bacilli + human leucocytes:
50 cells contained 149 bacilli.

One part of serum and two parts of an emulsion of human tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 60 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes:
50 cells contained 20 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes:
50 cells contained 56 bacilli.

One part of serum and two parts of an emulsion of avian tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 28 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes:
50 cells contained 12 bacilli.

OBSERVATION VII.—CASE OF PULMONARY TUBERCULOSIS WHICH HAD NOT BEEN TREATED WITH TUBERCULIN.

A.—Patient's serum + emulsion of human tuberculin bacilli + human leucocytes:
50 cells contained 80 bacilli.

Equal portions of the serum and an emulsion of human tubercle bacilli were digested for two hours at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 11 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes:
50 cells contained 20 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes:
50 cells contained 38 bacilli.

Equal portions of serum and an emulsion of avian tubercle bacilli were digested for two hours at 37° C. and then centrifuged at high speed.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes:
50 cells contained 18 bacilli.

OBSERVATION VIII.—CASE OF TUBERCULOUS MENINGITIS WHICH HAD NOT BEEN TREATED WITH TUBERCULIN.

A.—Patient's serum + emulsion of human tubercle bacilli + human leucocytes:
50 cells contained 136 bacilli.

Equal portions of the serum and an emulsion of human tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 60 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 29 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes: 50 cells contained 128 bacilli.

OBSERVATION IX.—CASE OF TUBERCULOUS CYSTITIS WHICH HAD BEEN TREATED WITH TUBERCULIN.

A.—Patient's serum + emulsion of human tubercle bacilli + human leucocytes: 50 cells contained 140 bacilli.

Equal portions of the serum and an emulsion of human tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 44 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 40 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes: 50 cells contained 120 bacilli.

Equal portions of the serum and an emulsion of avian tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 54 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 55 bacilli.

OBSERVATION X.—CASE OF TUBERCULOUS OSTEITIS WHICH HAD BEEN TREATED WITH KOCH'S TUBERCULIN.

A.—Patient's serum + emulsion of human tubercle bacilli + human leucocytes: 50 cells contained 150 bacilli.

Equal portions of the serum and an emulsion of human tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 44 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 20 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes: 50 cells contained 64 bacilli.

Equal portions of the serum and an emulsion of avian tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 33 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 20 bacilli.

OBSERVATION XI.—CASE OF TUBERCULOUS OSTEITIS WHICH HAD BEEN TREATED WITH KOCH'S TUBERCULIN.

A.—Patient's serum + emulsion of human tubercle bacilli + human leucocytes: 50 cells contained 135 bacilli.

Equal portions of the serum and an emulsion of human tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 53 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 49 bacilli.

B.—Patient's serum + emulsion of avian tubercle bacilli + human leucocytes:
50 cells contained 123 bacilli.

Equal portions of the serum and an emulsion of avian tubercle bacilli were digested for one hour at 37° C. and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes:
50 cells contained 58 bacilli.

(2) Equal portions of clear fluid + avian tubercle bacilli + human leucocytes:
50 cells contained 50 bacilli.

Summary of the foregoing Group of Observations upon the Amount of Phagocytosis that occurs when Human and Avian Tubercle Bacilli are presented to Normal Human Leucocytes in the Serum of Patients suffering from Tuberculosis, after Saturation of different Samples of this Serum with the Human and Avian Bacillus respectively.

The observations show uniformly, and in each case clearly, that the "opsonin" in the blood serum of a tuberculous patient is removed, or more correctly reduced, when the serum is saturated with the avian bacillus as markedly as when it is saturated with the human bacillus. No specific difference in the behaviour of the two bacilli is brought out by these observations. To cite one as a pronounced example: In Case 1, of pulmonary tuberculosis not treated with tuberculin, complete saturation of the patient's serum with human tubercle bacilli completely removed all the "opsonin" that can be regarded as specific, both for the human bacillus and for the avian; the phagocytosis fell from 135 in the unsaturated serum to 20 (human) and 34 (avian). And, as a corollary, saturation of the serum with avian bacilli reduced the phagocytosis of a suspension of avian bacilli from 320 to 51 (human) and 34 (avian). It may be pointed out that the number of bacilli in the human suspension used to test the phagocytosis and that in the avian was not equal, the suspensions having been made only approximately of the same density. The suspension of the avian was evidently somewhat stronger than the human. The fall, after saturation with the human, is from 135 to 20 (human) and 13 (avian). After saturation with the avian it is from 320 to 51 (human) and 34 (avian).

MISCELLANEOUS OBSERVATIONS UPON PHAGOCYTOSIS.

The following observation shows that in a bird suffering from active tuberculosis, the "opsonic" index is raised above the normal, both in regard to the human and to the avian bacillus.

The observation was made by using the leucocytes of a normal pigeon as phagocytes, in the serum of a tuberculous pheasant, and in that of a normal pigeon. The index in this particular observation is slightly higher in the case of the avian bacillus than in that of the human.

Observation XII.

In these experiments the serum was obtained from a tuberculous pheasant and the leucocytes from a healthy pigeon. The normal serum, which was used for control purposes, was also obtained from a healthy pigeon.

A.—(1) Fresh tuberculous bird's serum + human tubercle bacilli + bird's leucocytes were digested for twenty minutes at 40° C. 50 cells contained 100 bacilli.

(2) Normal bird's serum + human tubercle bacilli + bird's leucocytes: 50 cells contained 60 bacilli.

Opsonic index of tuberculous pheasant to human tubercle bacilli = 1·6.

B.—(1) Fresh tuberculous bird's serum + avian tubercle bacilli + bird's leucocytes were digested for twenty minutes at 40° C. 50 cells contained 44 bacilli.

(2) Normal bird's serum + avian tubercle bacilli + bird's leucocytes: 50 cells contained 20 bacilli.

Opsonic index of tuberculous pheasant to avian tubercle bacilli = 2·2.

The extreme difficulty of carrying out "opsonic" investigations with bird's leucocytes prevented us from making further observations with them; nor did we consider it important to do so in the face of the results obtained with human leucocytes and human serum. The difficulty arises in connection with washing the blood cells. If the blood is received directly from an artery into solution of citrate of sodium it clots, although under ordinary circumstances it remains unclotted. The blood of the bird does not coagulate at all when drawn straight from a vessel, provided that any contact with the injured tissues (other than the artery) is prevented, the injured tissues furnishing thrombokinase which, in the case of the bird, is not produced in the shed blood itself. We surmounted the difficulty by substituting ammonium oxalate solution for citrate of sodium. If the blood is received straight into this, clotting does not occur, and the corpuscles may be centrifuged and washed in the usual manner. Even then, the number of leucocytes which can be pipetted off from the surface of the washed blood cells is so small that the numeration of the bacilli in 50 cells is a task of much difficulty.

The following observations show that unless the serum of a tuberculous bird is used fresh no phagocytosis occurs when either human or avian tubercle bacilli are presented to the leucocytes of normal bird's blood.

Observation XIII.

In this case, the serum was obtained from six pigeons which had been inoculated with tuberculous material. The serum was forty hours old when these experiments were made, and had been allowed to remain in contact with the pigeons' red cells before centrifugalisation. The colour of the serum was golden yellow. The colour of the fresh serum as used in Experiment XII. was quite clear.

A.—(1) Serum from tuberculous (avian) pigeon + human tubercle bacilli + bird's leucocytes: 50 cells contained 6 bacilli.

(2) Serum from pigeon + avian tubercle bacilli + bird's leucocytes: 50 cells contained 6 bacilli.

B.—(1) Serum from tuberculous (avian) pigeon + human tubercle bacilli + bird's leucocytes:
50 cells contained 11 bacilli.

(2) Serum from pigeon + avian tubercle bacilli + bird's leucocytes:
50 cells contained 3 bacilli.

C.—(1) Serum from normal pigeon + human tubercle bacilli + bird's leucocytes:
50 cells contained 15 bacilli.

(2) Serum from pigeon + avian tubercle bacilli + bird's leucocytes:
50 cells contained 1 bacillus.

D.—(1) Serum from tuberculous (avian) pigeon + human tubercle bacilli + bird's leucocytes:
50 cells contained 3 bacilli.

(2) Serum from tuberculous (avian) pigeon + avian tubercle bacilli + bird's leucocytes:
50 cells contained 4 bacilli.

E.—(1) Serum from tuberculous (avian) pigeon + human tubercle bacilli + bird's leucocytes:
50 cells contained 12 bacilli.

(2) Serum from pigeon + avian tubercle bacilli + bird's leucocytes:
50 cells contained 2 bacilli.

F.—(1) Serum from tuberculous (avian) pigeon + human tubercle bacilli + bird's leucocytes:
50 cells contained 9 bacilli.

(2) Serum from pigeon + avian tubercle bacilli + bird's leucocytes:
50 cells contained 6 bacilli.

The following observation proves that the phagocytosis of normal human leucocytes is inhibited if human tubercle bacilli are presented to them in the blood serum of a normal pigeon.

Observation XIV.

(1) Fresh serum, obtained from a normal pigeon + human tubercle bacilli + normal human leucocytes: 50 cells contained 7 bacilli.

(2) Serum was obtained from a pigeon, and kept for fourteen days before being tested. Serum + human tubercle bacilli + normal human leucocytes:
50 cells contained 5 bacilli.

(3) Normal human serum + human tubercle bacilli + normal human leucocytes:
50 cells contained 225 bacilli.

The same emulsion of human tubercle bacilli was used in all these three experiments.

The following observation shows that the phagocytosis of normal human leucocytes is inhibited if either human or avian tubercle bacilli are presented to them in the serum of a tuberculous bird.

Observation XV.

The serum used in these experiments was obtained from a tuberculous bird.

A.—(1) Normal human serum + emulsion of human tubercle bacilli + human leucocytes:
50 cells contained 122 bacilli.

(2) Normal human serum + avian tubercle bacilli + human leucocytes:
50 cells contained 122 bacilli.

B.—(1) Tuberculous bird's serum + human tubercle bacilli + human leucocytes:
50 cells contained 41 bacilli.

(2) Tuberculous bird's serum + avian tubercle bacilli + human leucocytes:
50 cells contained 12 bacilli.

C.—The bird's serum was then digested for one hour at 37° C. with an equal portion of avian tubercle bacilli, and then centrifuged at high speed.

(1) Equal portions of the clear fluid thus obtained + human tubercle bacilli + human leucocytes: 50 cells contained 13 bacilli.

(2) Equal portions of the clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 14 bacilli.

D.—The same bird's serum was digested at 37° C., with an equal portion of human tubercle bacilli, and then centrifuged.

(1) Equal portions of the clear fluid thus obtained + human bacilli + human leucocytes: 50 cells contained 6 bacilli.

(2) Equal portions of the clear fluid + avian tubercle bacilli + human leucocytes: 50 cells contained 7 bacilli.

The fall in phagocytosis brought out in Observations XIV. and XV. is so striking that the bird's serum, whether normal or tubercular, must be held to exercise an inhibitive action upon the leucocytes of normal human blood. This appears from the circumstance that in the presence of salt solution simply, phagocytosis is not reduced to this extreme degree. For example: A suspension of *B. coli* in citrated salt solution *plus* washed normal leucocytes (from blood received into citrated salt solution and washed in salt solution) gave 223 bacilli in 50 cells. The same suspension of *B. coli*, *plus* the same washed normal leucocytes, without the use of any serum, gave 77 in 50 cells.

The following observations confirm the foregoing in showing that the phagocytosis of normal human leucocytes is inhibited in bird's serum, whether that of a normal bird or a tuberculous one, and whether human or avian bacilli are presented to the phagocytes or staphylococcus aureus. If the serum of the guinea-pig is substituted for the bird's, however, phagocytosis occurs when normal human leucocytes are used with it.

Observation XVI.

The birds' serum employed in these experiments was obtained from a normal pigeon and from a tuberculous pigeon which had been infected with the spleen of a tuberculous pheasant. The guinea-pig serum was obtained from an animal which had been inoculated with avian tubercle bacilli and which had completely recovered. The same emulsions of bacilli and cocci were used in all these experiments.

E.—Normal human serum + emulsion of human tubercle bacilli + human leucocytes: 50 cells contained 155 bacilli.

A.—(1) Normal bird's serum + emulsion of human tubercle bacilli + normal human leucocytes: 50 cells contained 20 bacilli.

(2) Normal bird's serum + avian tubercle bacilli + normal human leucocytes: 50 cells contained 20 bacilli.

B.—(1) Tuberculous bird's serum + human tubercle bacilli + normal human leucocytes: 50 cells contained 5 bacilli.

(2) Tuberculous bird's serum + avian tubercle bacilli + normal human leucocytes: 50 cells contained 7 bacilli.

C.—Normal bird's serum + emulsion of *Staphylococcus pyogenes aureus* + human leucocytes:
50 cells contained 23 cocci.

D.—Tuberculous bird's serum + emulsion of the *Staphylococcus aureus* + human leucocytes.
50 cells contained 23 cocci.

F.—Immune serum + emulsion of *Staphylococcus aureus* + human leucocytes:
50 cells contained 165 cocci.

G.—(1) Guinea-pig's serum + human tubercle bacilli + human leucocytes:
50 cells contained 145 bacilli.

(2) Guinea-pig's serum + avian tubercle bacilli + human leucocytes:
50 cells contained 56 bacilli.

In all the foregoing experiments the digestion was carried out for fifteen minutes at 37° C.

H.—Normal bird's serum + emulsion of *Staphylococcus aureus* + human leucocytes:
50 cells contained 18 cocci.

I.—Immune human serum + emulsion of *Staphylococcus aureus* + human leucocytes:
50 cells contained 198 cocci.

In the two previous experiments digestion was carried out for thirty minutes at 42° C., as approximating more nearly to the temperature normal to the bird.

J.—Normal bird's serum + emulsion of *Staphylococcus aureus* + human leucocytes:
50 cells contained 31 cocci.

In this experiment digestion was carried out for fifteen minutes at 42° C.

K.—Immune human serum + emulsion of *Staphylococcus aureus* + human leucocytes:
50 cells contained 107 cocci.

In this experiment digestion was carried out for fifteen minutes at 37° C.

GENERAL CONCLUSIONS.

(1) The human tubercle bacillus is pathogenic to the pigeon in a very limited degree only. It sets up no disease of the intestine, or of the abdominal viscera, when introduced (in sputum) with food into the alimentary tract, even for prolonged periods (six months), and in excessive quantities. It sets up a local or a local and glandular disease only, in the pigeon, when injected (in sputum) into the muscles or subcutaneous tissues. It sets up a local or a local and glandular disease only, in the pigeon, when injected into the muscles or subcutaneous tissues from the organs of guinea-pigs, which have been infected by inoculation with human sputum. The impossibility of producing infection in the pigeon by means of tuberculous sputum, as shown in our work, does not allow us to accept the statements which have been made in the other direction, for these statements do not rest upon experiment, but on inferences based only upon possibilities. Nocard's report of an outbreak of tuberculosis in a poultry-yard, where the man in charge was the subject of pulmonary tuberculosis we cannot regard as other than a coincidence. Avian tuberculosis may occur widely spread, where there is no evidence of infection from a human source. And

when intestinal ulceration occurs, as it does at times very extensively in fowls, the excreta would contaminate the soil, and furnish a ready means whereby the disease might be conveyed from bird to bird. Johne's examination of the livers of a number of fowls alleged to have been accidentally infected with phthisical sputum is open to the same criticism. Nocard himself failed to infect fowls by making them swallow tuberculous sputum, but he endeavours to explain away the result by stating that the condition of a laboratory and of a poultry-yard are altogether different. Although the disease may be spread from bird to bird, it has still to be determined whether there is any further or intermediate source of infection. Our experiments only show that if there is such, it is not human. Neither can we accept the statement, made by some, that the subcutaneous inoculation of birds with human sputum will produce avian tuberculosis, *i.e.*, the generalised and fatal disease as it is seen naturally in birds. In none of our pigeons has anything other than a local lesion, or a local and a glandular lesion followed; moreover, the disease so induced, instead of progressing, has (as told by the examination of birds at different dates after infection) retrogressed and, in some, disappeared.

(2) The avian tubercle bacillus, as tested from various kinds of birds, is pathogenic to the guinea-pig in a very limited degree. Here our results correspond with the previous statements made in this respect.

(3) The human bacillus has not, so far, in our hands, proved convertible into the avian by inoculation into the bird, *i.e.*, the bacilli have not proved identical. As illustrating the modification of a virus which may be brought about by its passage through animals of different species, the most striking example is still that furnished by human variola. And the possibility of a similar phenomenon has to be borne in mind in the case of the different forms of tuberculosis. Theoretically it would be quite possible that the human bacillus, after its insertion into the pigeon, might lose certain of its characters, and become incapable of exciting more than a trivial disease in the guinea-pig or in the human subject from which it was originally derived. This would be nothing more than what is witnessed in the results of human vaccination with calf lymph obtained by inoculating the calf with variolous pus.

Our experiments do not, however, support this theoretical possibility, the simplicity of which so strongly recommends it. Were it true, it should follow that the human bacillus, when introduced into the pigeon, should so lose its characters that when transferred from the avian lesions, or from the intact avian organs, to the guinea-pig, it should set up in this animal either no disease at all, or a modified form of disease, differing from that which arises after the direct inoculation with human sputum. Using pigeons, into the muscular and subcutaneous of which a salt emulsion of tuberculous sputum had been injected, we adopted the method of making a salt suspension of the spleen from these birds, killed at different dates after their inoculation (one week, two, four, six and eight weeks), and injecting this into the subcutaneous or muscular tissues of guinea-pigs. In addition, we similarly used the local lesion produced in the same pigeons. A fact of great importance to observe here is that the spleens of these pigeons in no case presented any naked-eye disease. In each bird there was a lesion at the site of inoculation ; in the birds allowed to live the longest this was of the smallest extent. Although there was no disease of the spleen, the salt suspensions of this organ, in every instance, produced not merely a local lesion in the guinea-pig, but a general tuberculosis.

In these experiments the human bacilli, thus transferred to the spleen of the pigeons from the site of inoculation, were placed in an ideal condition for the cells and juices of the bird to act directly upon them. Nevertheless the human bacillus, after a sojourn of as long as eight weeks in the spleen, produced the glandular and visceral disease in the guinea-pig as though it had been transferred directly to this animal from human sputum.[1] It did *not* produce merely a local, or a local and glandular disease, such as it should have done had it acquired the characters of the true avian micro-organism.

To these observations we may add a further, which has been already referred to under feeding experiments as showing the absence of tuberculosis in pigeons fed for prolonged periods with human sputum. A pigeon was fed for nineteen weeks with peas stirred in tuberculous sputum. The bird, when killed, presented no tubercular disease of the intestine microscopically, or of any of the viscera. Its spleen was rubbed up in salt solution and injected subcutaneously into a guinea-pig. The animal died in nine weeks, and showed a small local ulcer and much enlargement of the inguinal glands, together with disease of the spleen and scanty tubercles in the lung, as though it had received a direct inoculation with human sputum. It is not possible here to say how long the bacillus lay in the bird's spleen ; some time may have elapsed between the commencement of the feeding and the transference to the organ.

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[1] In a further experiment of this kind, the pigeon was killed three days after sputum inoculation (into the base of the wing); the injection of its spleen, in salt suspension, into a guinea-pig produced general tuberculosis, showing how rapidly the transference to that viscus is brought about.

In the determination of this question we think these results of greater cogency than those obtained by Nocard,[1] who attempted to test the action of the bird's fluids upon human bacilli by placing collodion capsules containing the human bacillus into the peritoneal cavity of the bird. Growing in such capsules the human bacillus became reduced in virulence, as might have been anticipated, seeing that it was forced to live in the toxic substances produced by its own metabolism—a result well recognised in the case of pathogenic bacilli grown under analogous circumstances. No such objection can be raised to our observations. It is significant, moreover, that the so-called avian bacillus thus evolved had not proved pathogenic to the bird on intraperitoneal injection, except in one instance; and that it would still kill guinea-pigs when injected intraperitoneally.

As evidence of the non-identity of the human and avian tubercle bacillus, we attach more importance to the positive results afforded by our feeding and inoculation experiments than to the negative evidence in the other direction furnished by the "opsonic" test. The latter shows, it is true, that either bacillus will extract the "opsonin" from tuberculous serum. But we do not regard this as a proof of their pathogenic identity; it is by no means a proof, *i.e.*, of the identity of their physiological or chemical properties. Certain investigations made by two of us (S. G. Shattock and L. S. Dudgeon) prove, amongst other things, that the "opsonin" may be very largely extracted from tuberculous serum by saturating it with a thick suspension of melanin—an observation showing that the removal of "opsonin" by bacilli is not wholly indicative of specificity, but that it is largely mechanical, the finely divided material entangling the "opsonin," and thus removing it from the centrifugalised serum.

The differentation amongst the members of any group of bacteria is not so much a morphological as a functional one, indicated by the chemical changes set up in the living body or in culture media. Where there is no disturbing factor introduced by conjugation or fertilisation, there can be practically no morphological variation, so long as the conditions to which the organism is exposed, remain constant. The differences amongst bacteria have probably arisen solely, or almost so, in consequence of "differences of environment." They are, therefore, relatively unstable. Even so definite a chemical condition as that which brings about the character of acid fastness is not fixed. The strain of avian tubercle bacillus, supplied from the laboratory of Král, is at present (in our subcultures) quite devoid of this character. And the same is true of our subcultures of the Timothy Grass bacillus from the same laboratory; although the subcultures retain their bright yellow colour and characteristic wrinkled habit, the micro-organism has completely lost the acid-fast quality.[1]

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[1] "Sur les Relations qui existent entre la Tuberculose humaine et la Tuberculose aviaire." Congrès pour l'Étude de la Tuberculose, 1898.

The determination of species amongst bacteria resolves itself, therefore, into one of stability; and the determination of the latter, into a question of time and environment. To fix any definite period, however, as a basis of such a definition, would be purely arbitrary and devoid of any scientific value. Whether the human and the avian bacilli have arisen from different stocks in diverse places, or arising from a single proximate source, have acquired their distinctive characters by prolonged transmission through particular classes of animals, we submit that the bacilli are at the present time sufficiently differentiated to rank as two separate micro-organisms. The difficulties surrounding the problem of their origin are the same as those which beset the source of difference between all allied plant or animal forms. There is in the case of the tubercle bacillus as much room for the monogenist and the polygenist as there is in the problem of anthropogenesis itself.

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[1] The bacillus was tested by the following methods:—(1) Three changes of hot carbol fuchsin, followed by 25 per cent. nitric acid. (2) Three changes of hot carbol fuchsin, followed by 5 per cent. nitric acid. (3) Hot carbol fuchsin, four minutes; saturated aqueous solution of picric acid and absolute alcohol, equal parts, three seconds; 5 per cent. nitric acid, three seconds; back to the picric acid and alcohol, three seconds.

Some Experiences with the Tuberculin Ophthalmic Reaction.

By L. J. Austin and Otto Grünbaum.

Any method which will assist in the diagnosis of tuberculosis with certainty would prove of extreme value to the human race, for there are many cases that cannot be diagnosed by the recognised clinical methods. The reaction following the injection of the old tuberculin of Koch may be applied in those cases in which the temperature does not oscillate through more than two degrees Fahrenheit, but in order to carry it out the patient must be confined to bed for several days, and the temperature recorded four-hourly. In addition to this, it is asserted that quiescent tuberculosis is occasionally awakened by the injection.

The estimation of the opsonic index, and the subsequent prolonged negative phase following the injection of tuberculin T.R., affords a harmless method of diagnosing the disease, but this necessitates the expenditure of so much time as to make it impracticable to carry it out on all cases that may be suspected of tuberculosis.

Von Pirquet [1], in September, 1906, published the results of some observations upon the development of papules after inoculating the skin with calves' lymph used for the ordinary Jennerian vaccination. He found that the size of the papule depended upon the dose of virus, and concluded that therefore it was produced directly by the virus and a property in the vaccinated which he termed "allergie." He believed that he had proved that the papule was not due to the multiplication of any micro-organisms, by the fact that the papules developed all about the same time, but varied in size. For instance: concentrated calf lymph resulted in a papule of 7·5 mm. after twenty-four hours, whilst if the lymph had been diluted to one in four the papule was 5 mm.; if one in sixteen, 3·5 mm.; if one in sixty-four, 2·5 mm.; if one in two hundred and ten, 1·5 mm.; and if one in a thousand, 1 mm.

In the following May, at a meeting of the Berliner Medizinische Gesellschaft, v. Pirquet [2] read a paper giving the results of a diagnostic method which he had devised for tuberculosis. The skin was scarified, and a 25 per cent. dilution of tuberculin was applied. In those cases suffering from tuberculosis a papule appeared within the next twenty-four hours. He came to the conclusion that the diagnostic value of this inoculation in young children was great, but that in later years all individuals reacted, and therefore it was valueless. Eighty-eight tuberculous cases gave a positive result, whilst fifteen cases of non-tuberculous disease failed to react. In no case was there any rise of temperature or any untoward signs.

Somewhat later, Moro and Doganoff [3] reported four cases of cutaneous tuberculosis inoculated by v. Pirquet's method, and in all the papule developed within twenty hours, but in three a phlyctenular conjunctivitis appeared about ten days later, occasionally lasting for a considerable period, whilst sometimes an exanthem made itself evident along with conjunctivitis.

Oppenheim [4] recorded similar observations, and came to the conclusion that patients suffering from cutaneous tuberculosis were remarkably sensitive to super-intoxication.

Abrami and Burnet [5] tried the cutaneous tuberculin reaction upon adults, and found that of twenty-one cases, none of which, so far as they knew, were suffering from tuberculosis, twelve reacted, whilst nine did not, and of these twelve, four very markedly. They therefore concluded that it is of no value upon the adult.

On June 17, 1907, Calmette [6] published a paper in *Comptes rendus de l'Académie de Science*, suggesting the transference of the v. Pirquet reaction from the skin to the conjunctiva, but in order to carry this out it was necessary to eliminate the effect of glycerin, with which ordinary tuberculin is usually prepared, and he used a 1 per cent. solution in sterile distilled water of a dried precipitate prepared by the addition of 95 per cent. alcohol to tuberculin. One drop of this was placed in the eye, and in tuberculous subjects redness and œdema appeared after five hours.

Breton and Petit had tried it upon a series of children, and found that sixteen tuberculous children reacted, whilst nine non-tuberculous children did not present any symptoms.

Calmette claimed that it was more rapid than the cutaneous method, and in the following month [7] refuted the statement by Vallée that the ophthalmic method caused too great an amount of pain to be of use to clinicians. At the same time he added that two new-born infants, whose mothers were tuberculous, failed to give the reaction.

In July, Calmette [8], Breton, Painblan, and Petit gave a summary of observations upon more than three hundred cases, but did not give details, and their conclusion, which is all that interests us, was that the ophthalmic reaction permits the diagnosis of tuberculosis with considerable certainty, even when clinical and bacteriological examinations throw but little light upon the case. They did not, however, apparently insist upon some facts which were brought out in a paper published by v. Pirquet [9] in September, based upon observations upon a hundred cases of children in which post-mortems had been obtained. Von Pirquet found that the cutaneous reaction, when obtained, was proof positive of the presence of tuberculosis, and that a negative result in the majority of cases meant an absence of tuberculosis, provided that it was carried out some time before death, for the test usually failed during the ten days previous to dissolution.

Letulle [10] tried the ophthalmic reaction on sixty-six subjects who were considered to be suffering definitely from pulmonary tuberculosis, and of these sixty-three gave a positive reaction, and three a negative reaction. He divided the reaction into three classes :—

(1) In which there was simple hyperæmia.

(2) In which there was marked hyperæmia without any muco-fibrinous exudation.

(3) Energetic reaction in which there was abundant exudation. Letulle concluded that it was not possible to tell to what extent there were pulmonary lesions by the intensity of the reaction.

Ferrand and Lemaine [11] compared the reliability of the cutaneous and ophthalmic reaction, and found that they did not agree. They tried the cutaneous reaction upon 100 cases; 46 were negative, and 54 positive. Of the 46 negative cases they subjected 17 to the ophthalmic reaction and obtained a positive result in 4, whilst of 54 positive cases they tried the ophthalmic reaction on 32, and only obtained it in 16.

Olmer and Terras [12] made a similar series of experiments, and they too found that the cutaneous and ophthalmic reaction did not agree. It is easy to understand that the cutaneous reaction is more difficult to carry out than the ophthalmic, for it is necessary to prevent inoculation of organisms which might lead to a reaction which could not be distinguished from that due to the tuberculin, but all that is necessary in the ophthalmic test is to be certain that there is no disease of the conjunctiva or the eyelids.

We have made observations upon some seventy-five cases, most of them believed to be suffering from some other disease than tuberculosis. The object of making the investigations was to see whether adults gave the ophthalmic reaction, for if a positive result was obtained, however slight the infection, and if Virchow's statement that we all suffer from a little tuberculosis were true, the reaction would be positive in all adults and therefore valueless.

In order to be able to study the reaction, its latent period, and the appearances that one had to look for, we carried out the test on some number of patients known to be tuberculous. The first thing that struck us was, that the five hours limit did not seem to be sufficient, some number of cases failing to present any alteration of the conjunctiva until nine hours after the drop was placed in the eye. It is well known that the development of the pustule after inoculation with calf lymph takes a longer time to develop in the winter than in the summer, and it is within the limits of possibility that the ophthalmic reaction occurs more rapidly in July than in November. We found that it was wise to examine the eye eight hours, twelve hours, and again twenty-four hours after the inoculation. With the exception of three cases, the reaction was mild, and did not cause any discomfort to the patients. It may be of interest to note that these three patients seemed to be cases who had an abnormally great resistance to the tubercle bacillus. The first was a man whom he believed free from tuberculosis; his complaint was gastritis, and he had become well under treatment. However, on making a detailed examination, we found there were scars on the neck, the history of which left but little doubt that they were of tuberculous origin. The second case was a man who was admitted for hæmoptysis suffering from tuberculosis. His first hæmoptysis occurred more than eleven years ago; when under our care he was well nourished, whilst his temperature remained within half a degree of 98° F. The third case was that of a girl, aged 13, who was admitted for tuberculous peritonitis. She improved so markedly under treatment that we were inclined to revise our diagnosis.

It is always dangerous to draw conclusions from a few cases, but it is nevertheless possible that patients possessing abnormal resistance give a more intense reaction than those in which resistance is low, whilst abnormally low resistance which must occur shortly before death may make the "allergie" approach *nil*, and under those conditions the test fails. Of the cases we tried, 21 proved positive, and we have little doubt that 20 out of these 21 were suffering from tuberculosis. The one in which we could not find evidence of infection had recently suffered from pyuria, but the urine did not contain any tubercle bacilli, and detailed examination failed to show any evidence of tuberculosis in the rest of the body; but it is necessary to make a detailed post-mortem examination in order to exclude the possibility of tuberculosis. Two cases in which we suspected tuberculosis failed to give reaction, but in one of these the evidence of tuberculosis was not very overwhelming.

It has been asserted by some that patients suffering from enteric fever give a positive reaction. We tried it in eight, and failed to obtain a positive result in any.

We tabulate our results, and conclude that the tuberculin ophthalmic reaction, from our small experience, has proved satisfactory in that the error seems to be small, and we can corroborate the observations of the various French investigators.

It cannot be expected to be infallible, but, if correct in 90 per cent. of cases, it will be of great assistance in diagnosing obscure infection.

	Positive	Negative
Pulmonary Tuberculosis	14	1
Tuberculous Peritonitis	3	—
Pleurisy with effusion	1	2
Pleurisy without effusion	2	1
Tuberculous Dactylitis	1	—
Bronchitis	—	17
Typhoid	—	8
Pneumonia	—	3
Acute Rheumatism	—	3
Empyema pneumococcic	—	2
Diabetes Mellitus	—	2
Chronic Arthritis	—	2
Chlorosis	—	2
Hysteria	—	2
Influenza	—	1
Phlebitis	—	1
Acute Nephritis	—	1
Chorea	—	1
Acute anterior Poliomyelitis	—	1
Neuroma in the neck	—	1
Bronchiectasis	—	1
Liver abscess	—	1
Infective Endocarditis	—	1

The single case of definite pulmonary tuberculosis in which a negative result was obtained was suffering mainly from a new growth pressing upon the spinal cord. The reaction was tried shortly before death, but in all probability the ophthalmic reaction will be found to be similar to the cutaneous reaction, in that a positive result will not occur during the last ten days.

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Abscess of Bone caused by an Intermediate Bacillus (P.) allied to B. paratyphosus.

By F. G. Bushnell, M.D.

E. T., aged 41, was admitted to the Sussex County Hospital, Brighton, under the care of Mr. R. F. Jowers, F.R.C.S., on May 25, 1907, for acute suppurative periostitis. There was a history of a mild attack of "typhoid" five weeks previously, and this illness came on during convalescence. On examination, there was a swelling the size of a bantam's egg about the centre and in front of the left tibia. This was tender, fluctuating, but not discoloured, and there was no sign of disease elsewhere. The temperature was 99·9° F. The abscess was incised on May 25, and about 100 cc. of thin pus was evacuated from beneath the periosteum; the bone beneath was not softened. A rubber band was afterwards applied above the knee for six hours daily. The wound healed rapidly and the patient was discharged well on June 19.

Dr. F. S. Beachcroft, of Petworth, writes that the patient was seen by him on April 13, and his appearance and symptoms led him to diagnose typhoid. There was a previous history of drinking tap water from the garden and also, about ten days previous to the onset of his illness, of eating whelks vended off a barrow. Other members of the patient's family had eaten these and were all ill immediately afterwards with diarrhœa and sickness, but they were well in a few days. The illness followed a typhoidal course, but there was diarrhœa only for the first five or six days. "Rose spots" were fairly abundant. About the twelfth or fourteenth day of his illness the blood gave a negative Widal's reaction. During convalescence, on May 18, pain was complained of in the ankle and in the lower part of the tibia, and the patient was sent to the County Hospital.

Bacteriological Report.

Pus from the abscess contained slender Gram negative bacilli. It was inoculated in broth, incubated at 37° C., and gave uniform turbidity without scum in twenty-four hours, due to the presence of the Gram negative motile bacillus in pure culture. On gelatine, incubated at 22° C., there were numerous small, transparent colonies with rounded or somewhat irregular borders, which did not alter appreciably in forty-eight hours and did not liquefy the gelatine. The bacillus measured 0·5 to 1 μ in width by 1-2 μ in length: on agar it grew as a moist, semi-transparent growth; it produced no spores, no pigment, and possessed no proteolytic powers; it fermented dextrose (glucose), maltose and mannite with gas formation, lactose was unaffected (with one doubtful exception in four inoculations), sucrose was unaffected but not rendered alkaline. The ratio of H to CO_2 formed was not estimated.

On two occasions milk was acidified in twenty-four hours and acidified and curdled in forty-eight hours, remaining so for three months. On one occasion an alkaline reaction was observed in three months in the litmus milk. There was no indol formed in peptone water in fourteen days. Shake culture of taurocholate neutral red agar showed yellowing for about 0·25 cm. from surface in 48 hours.

Cultural Characters.

	Levulose	Acid and gas bubble	Twenty-four hours at 37° C.
	Galactose	Acid and bubble of gas	
Monosaccharid	Dextrose	Acid and gas formed.	
Polysaccharid	Maltose	Acid and gas formed.	
	Sucrose	No acid or gas formed.	
	Lactose	No acid or gas formed (one doubtful exception on four occasions).	
	Raffinose	No acid or gas formed.	
Alcohol	Mannite	Acid and slight gas formed.	
	Dulcit	Acid and slight gas formed.	
Inversion product	Dextrin	Slight formation of acid and gas.	
	Inulin	No acid or gas.	
	Salicin	Negative.	
	Taurocholate Neutral red agar	No appreciable yellowing.	
	Milk	Acid and clot in forty-eight hours (alkaline on one occasion).	
	Peptone	No indol formation in fourteen days.	
Sodium taurocholate	Litmus glucose	Acid and slight gas.	

It was not tested on xylose or arabinose, but Ford states that the hog cholera group cannot ferment these carbohydrates, whereas the human parasitic *Bacilli icteroides* and *paratyphoid* and *B. enteritidis Gärtner* and swine dysentery can do so. The bacillus possessed considerable fermentative powers, as distinguished from the paracolon bacilli of the normal intestinal tract, which stand close to *B. fæcalis alkaligenes* in their low fermentative powers.

The paratyphoid group (known variously as paracolon, Gärtner, hog cholera, intermediate) are characterised by not coagulating milk, by producing no indol, by not fermenting lactose with gas formation, and not agglutinating typhoid serum, and by fermenting glucose with gas. As previously mentioned, two subgroups are formed by the reactions with xylose and arabinose according to Ford, and by their reactions with milk according to Buxton and others. Thus, with milk, cultures of typhoid become acid and remain so *permanently;* with paratyphoid, subgroup B, the ordinary form, milk shows an initial acidity, followed by alkalinity, in about two to ten days. The alkali formed is sufficient to dissolve or saponify the casein and clear the milk, or render it opalescent. It is possible the clearing is due to formation of casease by these bacilli. It is to be noted that the presence of fermentable galactose or glucose in milk would account for the permanent acidity of milk with typhoid or the initial acidity of paratyphoid. The rare paratyphoid subgroup A produces definite persistent acidity, but not enough to coagulate the milk. Buxton considers that the paratyphoids (causing typhoidal symptoms) are of two species, one (β) resembling the paracolons, and one unlike them culturally (a). The latter would be grouped under A.

The cultural characters were compared with paratyphoid a and β Buxton, with Schottmüller, Gärtner, and *B. coli communis.*

The serum reactions were very significant. The patient's serum, when under the care of Dr. Beachcroft and when examined by me on June 3 and 6, was negative to the laboratory culture of *B. typhi* in thirty minutes in 10 per cent. dilutions. It was positive in 1 per cent. dilution in thirty minutes to Schottmüller, to paratyphoid a/04 Buxton and Gärtner, but negative to *B. coli* (few small clumps in thirty minutes).

There was immediate agglutination and complete loss of motility in 1 per cent. dilutions of the patient's serum and his bacillus on April 29, on June 3, and on June 4. [No explanation is offered of the fact that it agglutinated with a typhoid serum (the eighth week positive).]

REMARKS.

The formation of a bone abscess due to paratyphoid infection is rare, and hitherto unrecorded to my knowledge, though paratyphoid fever is not uncommon in Sussex. There is presumptive evidence from the history, clinical account and absence of typhoid serum reaction, that the first illness was a paratyphoid attack. The bacillus isolated from the pus agglutinated the patient's blood serum and clearly caused the suppurative periostitis. As it coagulated and acidified milk and fermented glucose and maltose with gas, it was not typhoid, and lactose being practically unaffected, it was not *B. coli communis.* There is no doubt that a diagnosis of paratyphoid could have been arrived at if this bacillus had been employed in the first illness for the serum reaction. Keen, quoted by Dudgeon, states that *B. typhi* has been isolated from fifty-one cases of bone lesions following typhoid on thirty-eight occasions. Osler mentions six cases of bone lesions among the sequelæ of typhoid fever under his care in one year. Cushing isolated his bacillus O from an abscess following a typhoidal disease, and this bacillus was erratic in its production of alkali in milk. Castellani has isolated a bacillus with very similar characters from an appendicitis. I am indebted to Mr. H. N. Fletcher, house surgeon to the Sussex County Hospital, and to Dr. Beachcroft for the notes of the case, and to Mr. Jowers for access to the same.

The sudden onset of symptoms of poisoning after the eating of the shell-fish by other members of the family is comparable to meat poisoning by *B. enteritidis Gärtner*. In this patient invasion of the system by bacilli occurred.

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Pathological Section.

January 21, 1908.

Mr. S. G. Shattock, President of the Section, in the Chair.

A remarkable Case of Diffuse Cancellous Osteoma of the Femur following a Fracture, in which similar growths afterwards developed in connection with other bones.

By W. H. Battle and S. G. Shattock.

De femoris osteomate diffuso post fracturam evoluto.

SUMMARIUM.

Aeger, puer annorum quattuor de sede cecidit, femore suo sinistro in hoc modo fracto.

Solidata est fractura. Tumor autem apparebat (sarcoma osteoides ut videretur), et membrum amputatum est.

Membro in longitudinem secto, femur, extremitatibus suis exceptis, tumore osseo circumdari repertum est.

Parietis compacti axis aliquantulum devius fracturae locum indicat.

Tumor ex osse ubique constat cujus cancelli telâ adiposâ implentur.

Scrutatio microscopica telam tumoris osseam probat normalem esse.

Inter cellulas adiposas, locis in quibusdam, telae medullaris inseruntur tractus ex myelocytis cum granulis tenuibus constantes, ex myelocytis cum granulis crassis, ex lymphocytis, erythrocytis, deinde, inter has cellulas compressis.

Neque telae sarcomatis nec inflammationis adsunt notae.

Neoplasma itaque osteoma simplex ac benignum haberi debet.

Notandum est quod annum post unum hujus pueri matris membrum inferius amputatum est femoris adversus tumorem.

Hic tumor ex cartilagine in trabeculis imperfecte calcificatis dispositâ constat, cujus cancelli telam connexivam continent.

Cavitas femoris medullaris non occluditur.

Sarcomatis notis absentibus dubitari non potest quin neoplasma benignum sit. Chondromatis osteoidis, ut appellatur, in classe includendum est.

Osteomatis evolutio post femoris fracturam in puero, exemplum, igitur, fit hereditatis tumorum benignorum quae apud chondromata atque osteomata tam insignis et bene probata est.

In puero fracturam, opinamur, neoplasmatis causam excitantem fuisse, et neoplasma ipsum e callo reparante ortum esse.

In puero eodem, insuper, osteomata postea apud femur dextrum excreverunt, apud tibiam dextram, atque e radio et ulnâ.

Tumor qui portionem femoris dextri inferiorem amplexus est sua sponte disparuit.

E syphile haud aegrotaverat puer.

The following case is of so great rarity and, at the same time, of such interest, both clinically and pathologically, that we venture to think it worthy of a full and illustrated record. Clinically the lesion gave rise to an error of diagnosis, for the case was regarded as one of subperiosteal sarcoma, and upon this supposition the limb was disarticulated at the hip. Nor can it be said that this is the only form of lesion that has given rise to a similar error, and we may add to the clinical account of the case some examples in which a marked difficulty has attached to the diagnosis.

The patient, W. S., a boy, aged 4, was sent to St. Thomas's Hospital by Dr. Dowding, of Chatham, in 1901, for a tumour of the left femur. This presented some unusual characters, and he was shown to the Fellows of the Medical Society at one of the clinical meetings of that year.[1] He is stated to have been quite well up to the time of the following injury: he broke his left femur in April, 1901. The limb was put up in plaster of Paris, which was removed at the end of three weeks on account of excessive swelling of the thigh, which was said to have reached three times the size of the other one. The femur united, and the patient was able to walk about. The swelling was said to be diminishing.

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[1] See *Trans. Med. Soc. Lond.*, 1902, xxv., p. 327.

On examination the left femur was found to be the seat of a hard fusiform swelling, the thigh measuring 15 in. in circumference—twice the size of the right. There was no pain or tenderness, and the child appeared to be in good health, and got about without splints. No enlarged glands could be felt in the groin or any other part of the body. All the bones of the skeleton were examined by means of the X-rays, but nowhere could any abnormality be detected excepting in connection with the left femur.[1] Concerning this bone, the skiagraphic diagnosis made was periosteal sarcoma.

Amputation at the hip-joint was performed on November 8, 1901, by antero-posterior skin flaps, the abdominal aorta being compressed by the thumbs of an assistant. The muscles were divided down to the innominate bone. Very little blood was lost, and the operation was well borne. The patient was discharged from the hospital on January 18, 1902. His general health was then very good. The only other abnormality in the skeleton appeared to be a thickening of the left ulna below the elbow-joint; here there was some tenderness with limitation of the movements of the joint, and rotation of the radius was also impaired.

As regards the family history of the case, the patient was, at that time, the only child, and there was no history of tuberculosis or syphilis. It is, however, a remarkable fact that a few months later the boy's mother developed a tumour of her right femur, for which amputation of the thigh was performed by Mr. Cotman at the Rochester Hospital in 1903. The mother is, at the present date, alive and well, no recurrence of the tumour having taken place.

Mr. Pitcairn, the house surgeon at the Rochester Hospital, has, within the last few days, informed us that a younger brother of the boy, aged 2½, was admitted in July, 1907, with a fracture of the right femur a short way below the middle. The child was under care on account of the development of an extensive tumour of the thigh, the circumference of the limb at one time being 19 in. A portion of the growth was removed for microscopic examination, the report being that it consisted of well-formed bone, with connective tissue and cartilage in process of ossification.

On June 9, 1903, the boy whose case is described in the present communication was again brought to St. Thomas's Hospital on account of swelling on the right femur. This was situated at the lower part of

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[1] The various skiagrams referred to, and the reports upon them, were furnished by Dr. A. H. Greg.

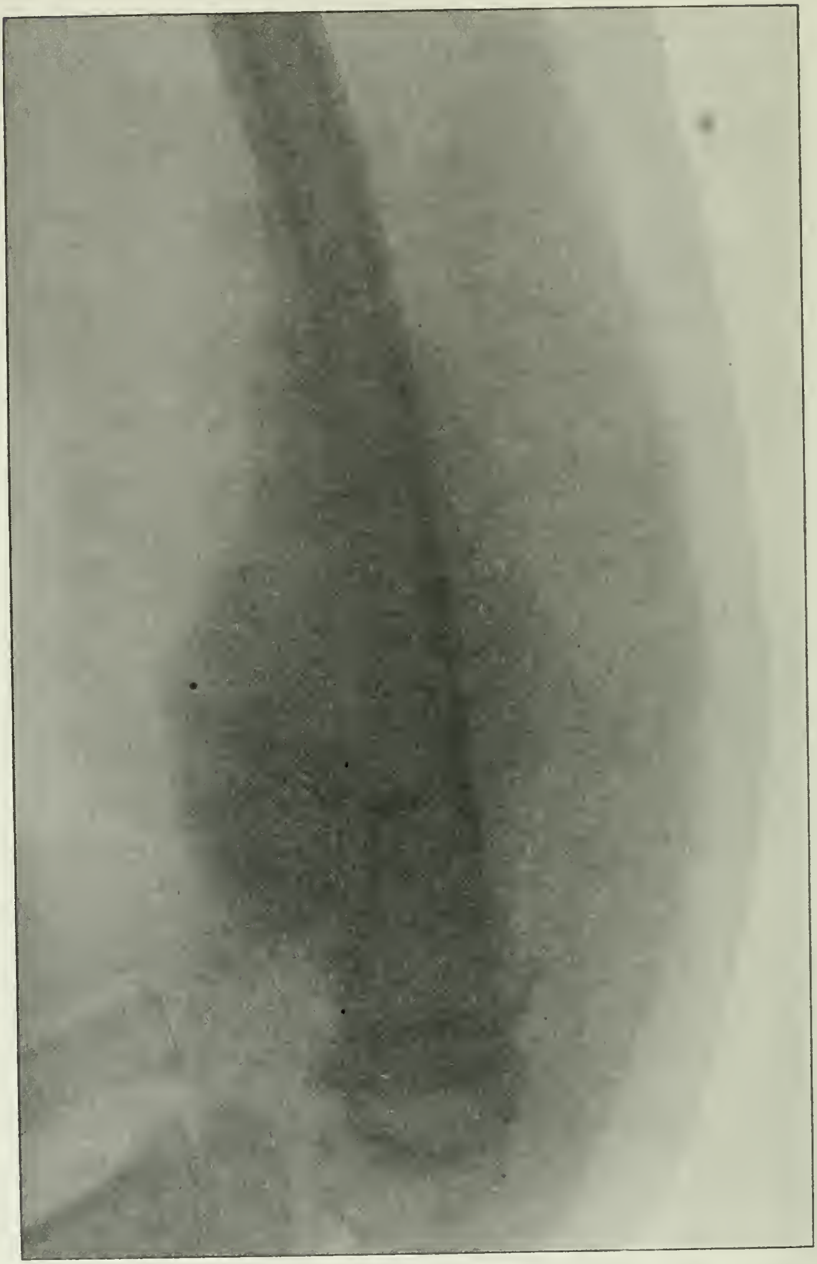

Fig. 1.

A skiagram of the right femur, showing a formation of bone surrounding the lower half of the shaft. When the limb was skiagraphed four years later the tumour was found to have disappeared.

Femoris dextri skiagramma. Osteoma portionem diaphysis inferiorem amplectitur. Tumor sua sponte disparuit.

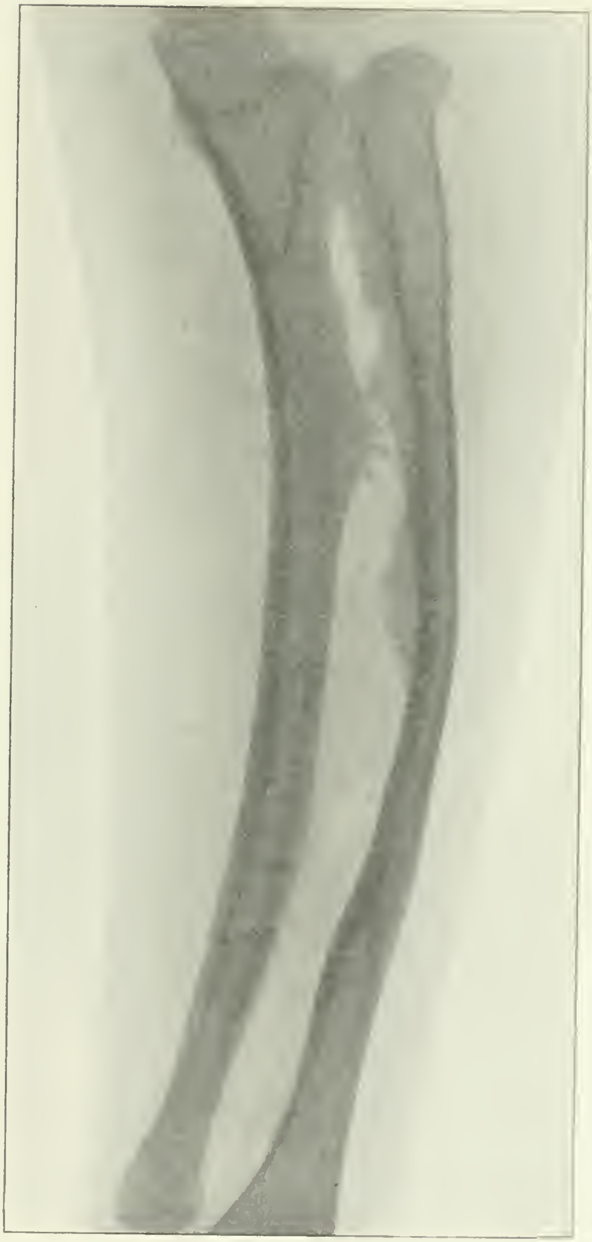

Fig. 2.

A skiagram of the radius and ulna. From each bone a flattened, irregular osteoma has grown, apparently in connection with the interosseous membrane.

Radii ulnaeque skiagramma. Ex utroque osse osteoma, in membranâ interosseâ, ut videtur, excrescit.

the shaft of the bone, and formed a rounded, hard, bone-like elevation the size of a large orange. It surrounded the lower part of the diaphysis. It was not particularly tender, and the patient could walk on the limb without pain, using a crutch. The disease was regarded as a sarcoma of the same kind as the original growth on the other femur. As it was thought that there were some faint indications of "lines" at the angle of the mouth, a course of hyd. cum cretâ was prescribed. The general condition was good, and he was active in his movements like an ordinary healthy child.

The boy was readmitted to the hospital on March 5, 1907, for renewed examination, as it was reported that he had developed a further tumour on his right leg. A year before he had hurt his right shin in a fall on the pavement. He was taken to a local hospital and admitted the following day on account of much pain in the shin and a swelling. An incision was made into the swelling and a diagnosis of sarcoma was made, but nothing further was done. The pain and the swelling afterwards diminished to some extent. On readmission his general health was good and he was bright and cheerful. The appearance of the patient had, however, altered considerably; the head had become larger and presented a somewhat peculiar appearance, being much widened across the frontal region, whilst it projected markedly in the occipital region, as if from large, fairly symmetrical bony prominences.

Report of the X-ray department, May 7, 1907: "The boss on the skull is a thickening of the outer table."

The forearms appear deformed, especially on the ulnar sides, where the bones project unduly below the elbow-joints. Both arms are abducted.

Report of X-ray department, May 7, 1907: "In the forearms the bones are deformed and present small osseous projections which show no characters of inflammation and have no resemblance to sarcoma. There is no evidence of fracture."

There was a large fusiform swelling of the tibia, hard to the touch, evidently osseous, and not at all tender. It measured 10½ in. in circumference at the widest part, and did not extend either to the knee- or ankle-joint.

Report of X-ray department, May 7, 1907: "The right tibia has a large osseous tumour on it: the bone, which is bent, can be just made out going through the tumour. The tumour has a skeleton of bone which is not arranged like that of an ordinary periosteal sarcoma; the outline, too, is circumscribed as in an osteoma. There is no appearance of active disease."

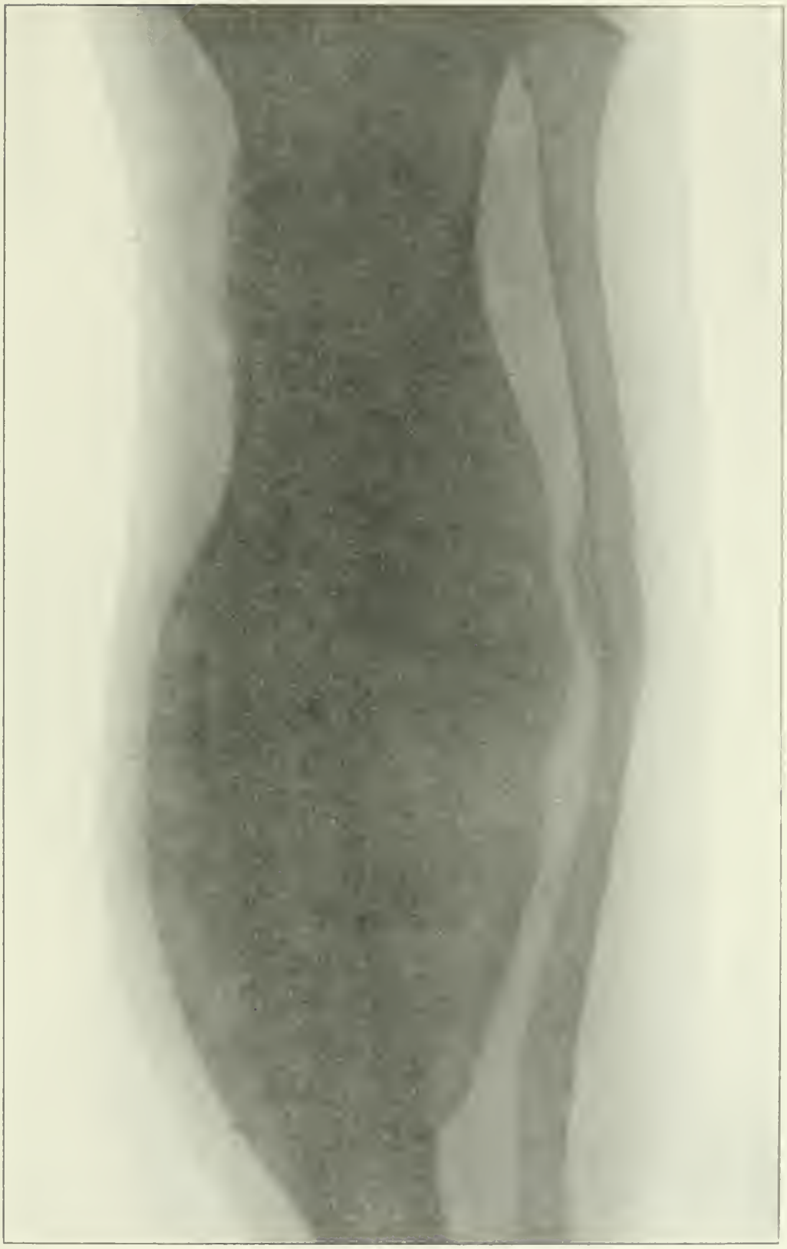

FIG. 3.

A skiagram of the right tibia, showing a fusiform formation of bone which appeared after a fall on the pavement. Slightly reduced.

Tibiae dextrae skiagramma. Diaphysis tumore osseo fusiformi circumdatur. Tumor post contusionem apparuit.

There were at this date no clinical signs of the previous tumour of the right femur.

Report of X-ray department, May 7, 1907: "The right femur (on which in 1903 there was a growth with all the appearances of a periosteal sarcoma) is now wide and abnormally transparent."

When this patient was shown at the meeting of the Medical Society no operation had been performed, and the opinion of the Fellows was asked for on the question of diagnosis. The following suggestions were made:—

(*a*) That the disease was a periosteal sarcoma of the femur, resulting from injury.

(*b*) That there had been a large subperiosteal extravasation of blood with subsequent ossification.

(*c*) That the swelling was a redundant growth of callus following the fracture of the bone.

On the whole, the suggestion which received most support was the one that the growth was a subperiosteal sarcoma. This was apparently confirmed by the result of the X-ray examination at a later date, and in accordance with this opinion amputation at the hip was performed. The pathological examination of the tumour, the subsequent appearance of other osseous growths of a non-malignant character, the disappearance of one of these under observation and the continued well-being of the patient prove this to have been an error.

A like mistake has been made on more than one occasion in regard to traumatic subperiosteal hæmatoma of a long bone, a lesion which has been known to arise in adult subjects who were otherwise in sound health. Needless to say, the disease for which the hæmatoma has been mistaken is the hæmorrhagic form of subperiosteal sarcoma.

Whether osteoma or subperiosteal hæmatoma following an injury, such cases so simulate the graver disease and so urgently suggest the propriety of amputation that the diagnosis becomes of the greatest importance, and it is a diagnosis which demands the highest acumen, both clinical and pathological, for its elucidation. Apart from cases of the second kind known to us indirectly or from a part-association in their diagnosis, there is in the Museum of St. Thomas's Hospital the femur of an infant showing such a hæmatoma, the case being one in which the error in question was made (No. 366 F). The specimen is a vertical section of the femur, from which the periosteum has been detached for the lower three-quarters, the separation extending inferiorly to the epiphysial line. The cavity before incision was filled with a clear, brown, viscid fluid. (The blood-clot now filling it is due to a recent hæmorrhage, which followed the exploration of the swelling.) On the front of the denuded shaft the space reaches a maximum depth from before backwards of 1·7 cm. and behind the shaft a depth of 2 cm.

In connexion with the periosteum which forms the outer wall of the cavity, a thin shell of new bone has been produced, and, as told by microscropic section, a notable amount of cartilage. The parts were removed after death from a child aged eight months, who was under the care of Dr. Coutts and Mr. C. S. Wallace.

The infant was breast fed for a week only, then fed with barley water and Nestlé's milk for five months, and, lastly, for three months with "Allenbury's" No. 3 Food. There was one other child in the family with marked rickets who had been fed in the same manner. For a month previously to admission to the East London Children's Hospital, the right leg had been tender, and for six days swollen and red. When admitted the thigh was found to be slightly swollen, shining and red. There were no abnormal signs in any of the other limbs. The swelling of the thigh extended, and as egg-shell crackling was obtained, an incision was carried down to the surface of the enlarged bone. This on being divided was found to be an osseous shell, about $\frac{1}{8}$ in. in thickness, enclosing a cavity filled with a clear, brown, viscid fluid; the shaft of the femur, bare of periosteum, was felt running through the middle of the space. The fluid proved sterile on culture. The general condition of the patient became steadily worse, and death took place on September 9, 1904, the child having been admitted on August 5, 1904.

In a paper elsewhere published, one of us (W. H. Battle[1]) has adduced further examples of the formation of swellings on the long bones (many of them following injury), in which the diagnosis from sarcoma may be difficult or even impossible. Of these perhaps the most common is excessive formation of callus after fracture. It is well known that undue movement of the fractured ends of a long bone will cause the formation of an excess of callus, but it is apparently in certain conditions of the nervous system that the largest formations are met with. Whether it is that owing to the nervous disease the parts involved are less sensitive to pain, and therefore are kept less quiet than in a normal individual, it is difficult to say. Possibly, owing to the loss of trophic influence, the formation of reparative material is not kept within proper bounds. Be this as it may, I (W. H. Battle) do not regard the result as in any way the direct consequence of the syphilitic infection which caused the disease for which the patient was under care at the time of the accident; that is to say, the swelling is not the result of a syphilitic osteitis, nor due to the formation of gummata. In one case mentioned (loc. cit.) the patient was suffering from locomotor ataxy. She was a very stout woman, aged 35, whose femur snapped at the neck as she was walking along a road. The patient was admitted forty-five days after the injury, during which time she had been kept in bed with a long outside splint applied. The limb was shortened 1 in. and a large bony swelling occupied the upper fifth of the femur, interfering with the action of the hip-joint. X-ray examination showed this to be osseous in nature and to be associated with a fracture of the neck of the femur. The swelling diminished slowly, being still very evident upon examination two years later.

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[1] *Lancet*, 1904, ii., p. 580.

One of us (W. H. Battle) has under observation at the present time a man, aged 45, who has had symptoms of general paralysis of the insane for about three years. This man fell in the street and sustained a fracture of the left femur in November of last year. The fracture was in the upper third of the shaft, and the superior fragment was very sharp. The patient on more than one occasion took off his bandages and splints and attempted to get up. When seen a month after the accident, there was much shortening and eversion of the limb, whilst the thigh was occupied by a great swelling which made it several inches larger than the right and suggested the presence of a sarcomatous growth. It was stated that this extensive swelling had only appeared a month after the accident. He had had very little pain, and his general health appeared good. X-ray examination, five weeks after the injury, showed the tumour to be an excessive formation of callus with ossification in the neighbourhood of the fracture. His reflexes were normal, but there was some increased frequency of micturition.

When osteitis deformans attacks one bone of the skeleton only, especially where there is a history of injury, some considerable difficulty may be experienced in coming to a correct conclusion. One of the cases mentioned (loc. supra cit.) was that of a married woman, aged 50, who was sent into St. Thomas's Hospital for a swelling of the right hip, which she had noticed for six weeks. A shortening of the limb with inward rotation caused inquiries to be made into her past history. It was found that she had worn a high boot for two years, ever since a fall in her kitchen. The limb was 2½ in. shorter than the other and the hip was considerably enlarged. X-ray examination showed that she had sustained a fracture of the neck of the femur, the upper part of the shaft, with the trochanters, being very much thickened by osseous formation. The bone was trephined; the microscopical appearance of the piece removed "suggested a chronic osteitis." Under the X-rays the arrangement of the bone resembled that seen in osteitis deformans. Her reflexes were normal, and there was no evidence of disease elsewhere, although the patient was an emaciated, feeble woman.

In the same paper (loc. cit.) a photograph is reproduced of the thigh of a young man with a swelling of the posterior part. The case was sent up as a doubtful one of sarcoma, but the shortening of the limb, the way in which the swelling shaded off on to the bone above and below, together with the result of X-ray examination, made the diagnosis of osteitis deformans certain.

Another condition giving rise to a swelling which simulates sarcoma of a long bone is quiet necrosis. The amount of enlargement here may be quite out of proportion to the extent of bone which has undergone necrosis. The sequestrum, indeed, is in some instances so small that even the use of the X-rays has not proved sufficient to clear up the nature of the disease and prevent the removal of the limb.

So far as we are aware, the only instance on record of the condition known as leontiasis ossea affecting other than the bones of the head is to be found in the *Transactions of the Pathological Society of London*. Here the fibula and the hyoid bone were affected, as well as the bones of the skull and face. The case is perhaps the most pronounced that has yet been observed, and is recorded (with plates) in the *Transactions of the Pathological Society*, 1865, xvii., p. 243, by Dr. Charles Murchison, the patient having been under the care of Mr. Bickersteth, of Liverpool.[1]

The patient was a man, aged 34, in whom enlargement of the bones of the face was first noticed when he was aged 14. Thirteen years after this a similar hard swelling appeared along the course of the left fibula. As the facial enlargement increased, the cavities of the mouth and nose were greatly lessened, and the eyeballs extruded. Neither the integuments nor the soft parts were implicated in the disease. The patient had not suffered from syphilis, either congenital or acquired. One brother had a similar enlargement affecting the upper jaw on one side; this growth had also commenced about the age of puberty, but had for many years remained stationary.

After death no visceral disease was found. The bones affected were those of the face and cranium, the hyoid, and the left fibula. The disease of the skull consists in great thickening and induration, the condition being due chiefly to the growth from the outer surface of numerous, closely aggregated, smooth, dense, botryoidal excrescences, varying in size from a hempseed to a small cherry. The only bone of the skull not involved is the occipital. The lower jaw presents the most remarkable change. It is enormously thickened in every direction; little trace remains of the condyles, coronoid processes or sigmoid notches, the whole being converted into two huge bilateral globular masses. The hyoid bone partakes in the same process, the body being much thickened and raised in botryoidal eminences. The shaft of the fibula, as seen in transverse section, is enlarged and rarefied in texture; the medullary canal is not occluded.

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[1] A plaster cast of this extraordinary specimen was recently presented to the Museum of the Royal College of Surgeons by Prof. A. Grünbaum.

From the posterior and internal aspect there springs a great overhanging bony mass, $4\frac{3}{4}$ in. × $5\frac{1}{2}$ in. in its chief diameters. The greater part of this osseous mass is made up of dense bone, with here and there an extremely delicate cancellous structure; the dense bone, on close examination, is seen to be perforated with numerous minute apertures.

A case of localised enlargement of the tibia, which is interesting in connection with the subject of our paper, will also be found in the *Transactions of the Pathological Society of London*, xxxvi., p. 388. There is, however, no definite proof that the patient had sustained a fracture of the bone. The case was reported by Mr. Bilton Pollard, and is discussed later on in the present communication.

PATHOLOGICAL ACCOUNT AND PATHOLOGICAL REMARKS.

By S. G. Shattock.

The nature of the condition will, perhaps, be most readily understood from the description of the specimen which I have drawn up for the Pathological Catalogue of the Museum of the Royal College of Surgeons, to which it has been added. The inner half of the left thigh and upper portion of the leg of a child, the parts having been divided sagittally. The entire limb was removed by amputation for the tumour shown, which appeared after the femur had been fractured by a fall. In the surface of the section, opposite the middle of the shaft of the bone, there is an indication of the fracture in a slight irregularity in the line of the proper compact wall, and in an interruption of the medullary canal.

The fracture has healed without displacement. In connection with it there has been produced a large tumour, the extent and disposition of which at first suggests its being a subperiosteal sarcoma. The new formation is most prominent on the anterior aspect, when it attains a maximum thickness of 4·5 cm.; posteriorly, where thickest, viz., opposite the site of the fracture, it does not exceed 2 cm. It involves, on both aspects, almost the entire length of the shaft, although on the front the volume of the growth is some five times or so that behind the bone. The anterior portion of the neoplasm presents itself as a hemi-elliptical mass intimately incorporated with the compact wall of the shaft, except inferiorly, where for a distance of 1·5 cm. it overlaps the anterior surface of the femur without being continuous with it.

Above the position last referred to the growth, for a short distance, is superficially lobulated, the structure being here penetrated by narrow septa of connective and muscular tissue. The highest part presents a somewhat similar configuration. Posteriorly the most prominent portion of the tumour corresponds with the plane of fracture, where it is continued downwards as a superficially lobulated sheath of diminishing thickness, almost as far as the epiphysial line. Above the plane of fracture it is represented by an investing process not more at first than 0·5 cm. in thickness, which becomes more and more attenuated as followed upwards, and terminates 0·4 cm. below the summit of the great trochanter. The compact wall of the shaft of the femur is traceable throughout beneath the new formation on both aspects, although its proper surface has lost some of its natural sharpness from a certain degree of superficial involvement.

There is nowhere any trace of similar growth in the medullary canal, which is occupied with adipose marrow. The growth presents but little vascularity, the most vascular area being that immediately behind the seat of fracture. To the naked eye, its structure is uniform throughout, and consists of finely cancellated osseous tissue, the interstices of which are filled with fatty medulla. If its texture be compared with that, say, of the upper end of the shaft of the tibia, no difference is discernible beyond that of the disposition of the cancelli in the two positions. There is nowhere the slightest trace, even at the surface, of a sarcomatous formation. The growth, from a naked-eye scrutiny, must be classed as an osteoma. A portion of the tumour, including the free surface, displays after maceration and subsequent treatment with ether a uniform somewhat finely cancellous structure, the trabeculæ of which are everywhere normally calcified.

A skiagram was taken of the preparation in order to see whether it would give any clue as to the nature of the growth. In its general form and disposition the tumour could not be diagnosed by this means from a subperiosteal sarcoma. Nevertheless, with the knowledge gained by the examination of the specimen, it is not difficult to recognise the presence of finely cancellous bone throughout the swelling, the texture being practically identical with that in the cancellous ends of the femur itself. Less clearly these appearances were indicated in the skiagram of the tumour taken before the removal of the limb. In this it was recognised at the time that the textural picture of the growth was unusual and that it differed from that of an ordinary osteoid sarcoma.

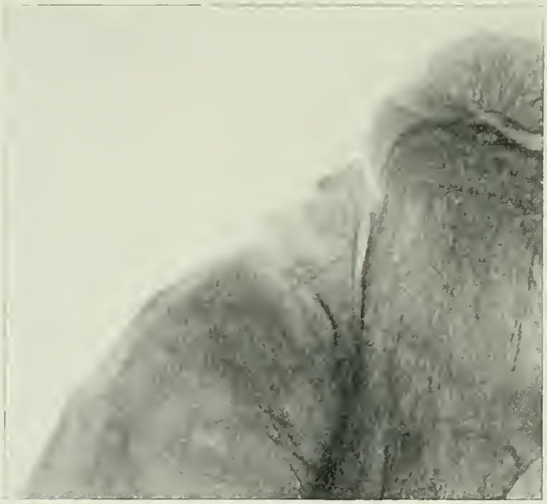

Fig. 4.

Portion of a skiagram which was taken from the bisected limb after preservation by the formalin method. The area selected is from the higher end of the growth, and includes the adjacent part of the femur. Throughout the new formation a delicate cancellous structure is discernible, even at its very surface, the texture being practically identical with that in the cancellous end of the femur itself. Natural size.

Membri skiagrammatis portio, femoris extremitate superiori inclusâ. Neoplasma ubique monstrat telae structuram osseae cujus cancelli haud illis dissimiles sunt qui femoris ipsius extremitatem constituunt. Magnitudinis naturalis.

Histology.—Both frozen and paraffin sections were made, a portion of the growth including the surface and overlying muscle being selected for examination. Decalcification was carried out by means of phloroglucin and nitric acid. For the study of the proper medullary elements Ehrlich's tri-acid and Leishman's stains were used. The structures displayed consist of normal adipose tissue in which lie slender osseous trabeculæ of equally normal character.

(1) To take the osseous constituent first. The trabeculæ comprise a uniform matrix and included corpuscles. In some portions of their extent certain of them are invested with a single series of osteoblasts, all the normal stages of cell inclusion in the matrix being obvious. The presence of canalicular systems in the matrix in connection with the lacunæ completes the characteristic normal structure. Here and there a group of osteoclasts lies against some portion of a trabecula, which is correspondingly eroded. Where the trabeculæ are not invested with osteoblasts the osseous substance lies in immediate contiguity with the fat. The actual surface of the growth is formed by a fibrous membrane with which the suprajacent muscular tissue is intimately incorporated.

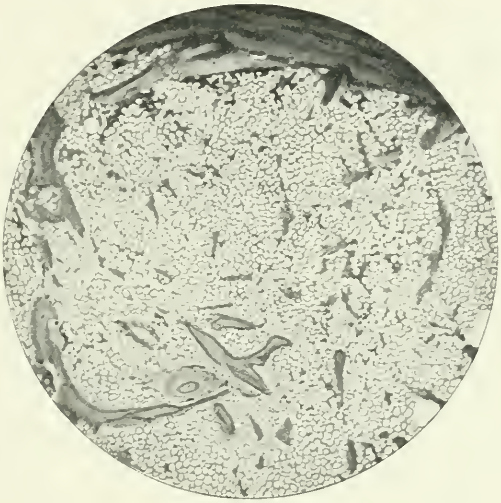

FIG. 5.

A microscopic section of the growth, including portion of the overlying muscle. It is constructed throughout of cancellous tissue, the spaces of which are filled with medulla, chiefly adipose tissue. Hæmatoxylin and eosin ; 2 in. objective.

Neoplasmatis sectio microscopica, musculi suprajacentis portione inclusâ. Ex telâ osseâ normali ubique constat cujus cancelli medullâ, praecipue telâ adiposâ implentur.

The overlying muscle presents itself as longitudinally disposed fasciculi of normally striated fibres with intervening zones or strata of fibrous tissue; in the immediate neighbourhood of the growth the muscular bundles cease, a similar form of connective tissue to that distributed between the fasciculi completing the limit of the tumour, with the difference, however, that it is here more richly provided with cells. The adipose tissue filling the intervals between the ultimate or most superficial series of trabeculæ lies in actual contact with this capsular

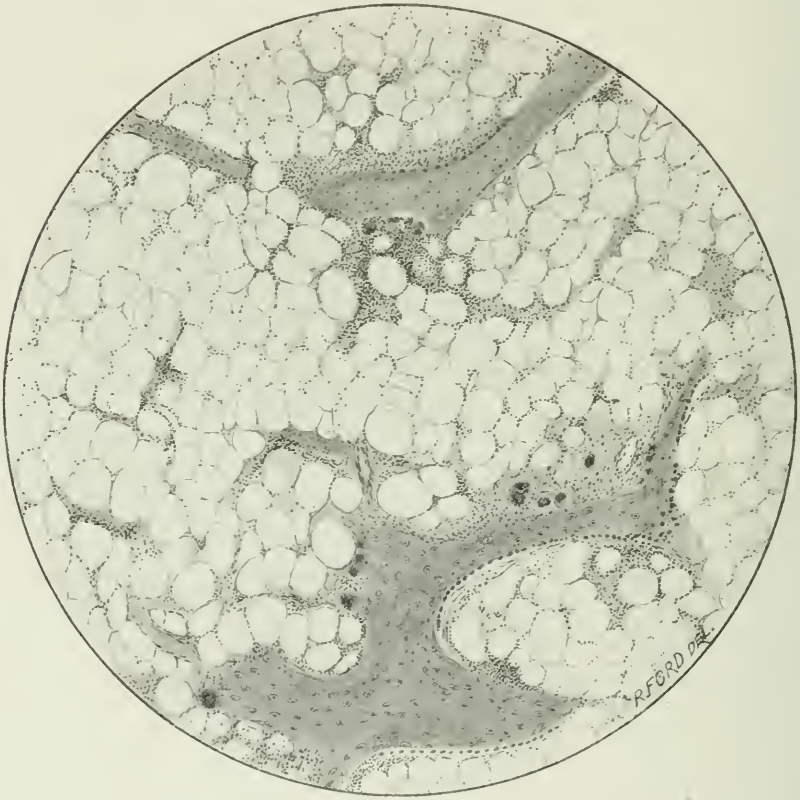

Fig. 6.

Portion of the section shown in fig. 5, somewhat more highly magnified. Two trabeculæ of normally formed bone are shown; these are in places invested with osteoblasts and in places eroded by osteoclasts. The intervening material consists of adipose tissue, in which strands of smaller-celled true medullary tissue occur. Zeiss obj. A, Oc. 4.

Neoplasmatis sectio microscopica. Ex telâ osseâ normali ubique constat cujus cancelli medullâ, praecipue telâ adiposâ, implentur. Inter cellulas adiposas inseruntur telae tractus medullaris verae.

membrane, that is to say, there are no limiting or circumferential laminæ of bone, and although a trabecula here and there meets the membrane obliquely, they mostly impinge upon its under surface in a vertical, or almost vertical, direction. At the sites of impingement the deepest, more cellular zone of the capsule is prolonged inwards upon the trabeculæ. The formation of bone must therefore be ascribed to this cellular or formative zone, which answers to the deeper layer of the

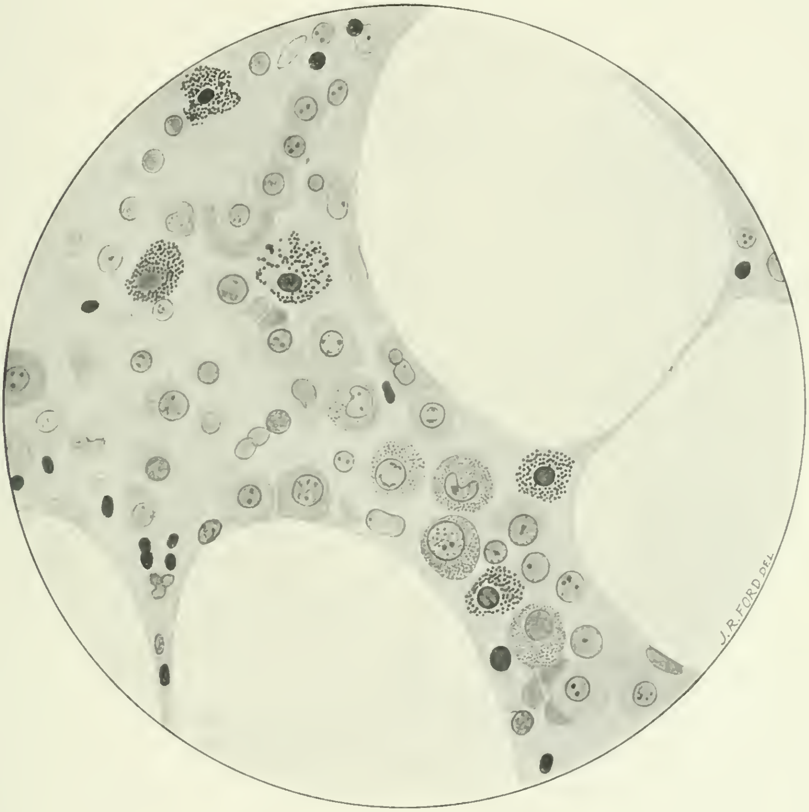

FIG. 7.

A microscopic section from the same situation as the preceding, as studied with $\frac{1}{12}$ oil immersion; Leishman's stain. Between the large vesicles of fat there is shown a strand of proper medullary tissue. In this may be recognised finely granular myelocytes with a single spherical or oval nucleus, lymphocytes, coarsely granular eosinophile or fuchsinophile myelocytes; and here and there a red corpuscle, intercalated and misshapen, amongst the other elements.

Sectio microscopica liquore Leishman tincta; $\frac{1}{12}$ obj. Inter cellulas adiposas in telae medullaris tractu discernuntur myelocyti cum granulis tenuibus, lymphocyti, myelocyti cum granulis crassis, et inter has cellulas erythrocyti interpositi.

periosteum. Indeed, as there is nothing between the surface of the shaft and the surface of the growth, the membrane investing the latter cannot but represent the periosteum.

(2) *The Medulla.*—By far the chief area of the section consists of ordinary adipose tissue, throughout which the osseous trabeculæ are uniformly distributed. The most interesting feature, however, is the presence of narrow strands of proper medullary tissue which track between the fat cells in different positions. In places they are quite absent, and they are most pronounced in the immediate neighbourhood of the periosteum. The proportion of true medullary tissue to the fat, however, is quite a low one. On a critical study all the forms of cell that make up the red marrow are recognisable. The chief number consist of finely granular myelocytes (with a simple spherical or oval nucleus) and of lymphocytes, and in conspicuous numbers there occur coarsely granular eosinophile or fuchsinophile myelocytes, the other elements in a given group consisting of erythrocytes, packed amongst and distorted by the other cells; here and there a nucleated red cell or erythroblast is encountered, or a polymorphonuclear leucocyte.

This remarkable new formation, distinctly referable to the injury, the fracture, and consisting throughout of well-formed cancellous bone with appertaining true medullary tissue, must be classified as an osteoma. It is an osteoma that has arisen, in its initial stage, as a callus formation, which, for some as yet inexplicable reason, has transgressed the normal bounds and exhibits all the physiological attributes of a tumour. Without the invasiveness, which is the cardinal mark of malignancy, it displays all the independence or anarchy of growth which characterises a benign tumour. It presents none of the histological marks of inflammation, and we must altogether discard the view of its being an inflammatory production, syphilitic or other. The growth of such an osteoma ensuing upon a fracture is certainly as rare as that of a sarcoma arising under the same circumstances, if it is not more so. One of us (S. G. Shattock) has discussed the latter sequence in recording an example of it.[1] The growth in this case arose at the site of a fracture of the humerus and occurred in a policeman who broke his arm in a heavy fall against an iron railing; this took place four and a half months before his admission into St. Thomas's Hospital. Splints had been kept on for five and a half weeks; when these were removed the arm felt normal, and union was complete. A month and a half after the discontinuance of the splints the patient noticed for the first time a lump in the region of the fracture; this increased somewhat rapidly afterwards, and for a while was regarded as an excessive production of callus. Recovery ensued after amputation at the shoulder-joint, but death took place about two years later with symptoms of recurrence in the chest. The rarity of such a sequence was fully recognised by Virchow, who had himself the opportunity of examining only one example, viz., a case in which Langenbeck had disarticulated the arm of a man, aged 23, for a growth which had appeared a year and a half after a fracture. The large proportion of cartilage in the microscopic sections from the case recorded in the *Transactions of the Pathological Society* is, as was therein pointed out, noteworthy in connection with the exciting cause of the growth; it relates the new formation to the reparative callus, which, as is now generally known, in simple fractures, in the case of the human subject as in that of the lower animals, undergoes a certain amount of cartilaginous metaplasia.

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[1] *Trans. Path. Soc.*, 1896, xlvii., p. 261.

In the *Transactions of the Pathological Society*, 1885, xxxvi., p. 388, Mr. Bilton Pollard has described under the title of "Hypertrophied Callus of the Tibia and Fibula" the case of a patient who, when a year old, fell off a chair and injured her left leg so as to disable her for a fortnight. Whether the leg was then broken or not could not afterwards be discovered. Nothing further was noticed until a year and a half after the accident, when the leg was found to be swelling in the middle; it was not painful, and locomotion was not interfered with. When the patient was admitted into University College Hospital, under the care of Mr. Marcus Beck, she presented a considerable enlargement of the middle of the tibia and there was a second swelling on the fibula. The tumour was supposed to be an ossifying sarcoma, and the leg was amputated. When the tibia was afterwards sawn down it was found to be the seat of a fusiform growth which microscopically presented a basis of cellular connective tissue, through which spicules of normal bone were evenly distributed. The macroscopic figure accompanying the paper shows the whole of the growth to be of a uniformly cancellous structure. The fibula was the seat of a similar but less extensive formation. Nevertheless, this case of Pollard's differs from the present in two important particulars: (1) the original shaft has been completely replaced by the new formation, which also fills the medullary canal; (2) the histological picture is markedly different, for the interstices of the cancellous tissue are not filled with medulla, either adipose or other. The attempt might be made to explain away the macroscopic dissimilarity by assuming that the new growth has arisen in connection with the internal or intramedullary callus as well as with the external or subperiosteal, and that the original compact wall of the shaft has not been invaded by any neoplasm (as it would be by a sarcoma), but that in the ordinary process of growth the shaft has been removed, and instead of being renewed it has been replaced, and come to be represented by the abnormal tissue, somewhat as in the progress of rickets, where the compact wall of the shaft of a bone, without being itself diseased, is removed *pari passu* with an excessive production of finely cancellous and imperfectly calcified bone of periosteal source.

In the case of the rachitic calvaria, the tables, like the compact wall of the long bones, disappear in the progress of the disease, the general texture of the skull becoming finely porous throughout and in macroscopic characters like that seen in the presclerotic stage of osteitis deformans, with this difference, however, that in the rachitic skull the trabeculæ are so imperfectly calcified that microscopic sections may readily be cut without any artificial decalcification. In discussing these changes[1] I have advocated the view that the osseous overgrowth occurring in rickets should be regarded as inflammatory.

With Mr. Bernard Pitts I have described at length[2] a remarkable case in which the upper two-thirds of the tibia of an adult, a woman, aged 37, was completely replaced by a non-calcified, finely cancellous bone; on careful inquiry syphilis, whether congenital or acquired, was in this case excluded. I did not regard the lesion as any form of sarcoma, and as it could not be identified with any condition hitherto described, I ventured to give it a distinctive name, viz., "non-calcifying plastic osteitis." It may be mentioned, in passing, as corroborative of the diagnosis against sarcoma that, although the amputation, which was carried out just above the knee, was performed in November, 1895, the patient, when seen three years later, was free of recurrence, and that she died of pulmonary disease about five years ago. The material filling the spaces of the non-calcified bone was not true marrow, but a very cellular connective tissue, without fat cells or myelocytes.

In regard, therefore, to Pollard's specimen I am disposed to agree with those members of the Morbid Growths Committee (which reported upon it) who viewed the lesion as a hyperostosis referable to an inflammatory process rather than an overgrowth of callus. The complete disappearance of the wall of the shaft and its replacement by the new bone, together with the extensive filling of the medullary canal and the absence of medullary tissue from the interstices of the new bone, seems to me to relate the formation to an inflammatory process and to displace it from the group of true osteomata. I know of no instance in which, in the case of a simple fracture, the wall of the shaft has vanished in the callus: and against the view, moreover, of an excessive callus formation must be placed the fact that the enlargement was not observed until a year and a half after the accident, whatever the nature of this may have been. An examination of Pollard's specimen (No. 1241 D, College of Surgeons Museum) leads me further to regard it as of the same character as that already referred to and described by myself in the tibia. As in the latter case so in the other, the new tissue is demarcated almost abruptly from the normal cancellous tissue of the tibia above and below, being mapped out by its abnormally fine texture and paler colour; and what makes the likeness still nearer is the circumstance that the new bone in Pollard's case is imperfectly calcified, though not to the extreme degree shown in the other.

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[1] *Trans. Path. Soc.*, 1891., xlii., p. 235.

[2] *Trans. Path. Soc.*, 1897, xlviii., p. 176. The specimen is preserved in the Museum of St. Thomas's Hospital.

Pollard's specimen I have re-examined microscopically by means of frozen sections cut vertically (without artificial decalcification) from the most prominent part of the tibial enlargement. The histological picture comprises well-formed trabeculæ (and included corpuscles) lined with osteoblasts; osteoclasts occur here and there. Between the trabeculæ there is throughout a young connective tissue rich in elongated cells, and with an imperfect indication of fascicular arrangement; the cells are separated in different degrees in different places by delicate intervening fibrillæ; nowhere are there any proper myeloid elements, and there is not a single fat cell. The trabeculæ are mostly unstained, refractive and calcified; some present an eosin-stained uncalcified margin.

There is no macroscopic trace at either epiphysial line of the changes pathognomonic of rickets, and this disease may be excluded. The chief difference between the two specimens is that in the adult tibia the new bone has replaced the old and filled the medullary canal *without producing any external swelling.* The imperfectly calcified and finely porous bone in both cases is much like that found in progressing rickets. In rickets, as in the other cases, I should regard the anatomical lesion as of the same nature, *i.e.*, an inflammatory formation of bone, in which imperfect calcification has occurred; the name already used in connection with the lesion of the tibia would apply to rickets—the lesions affecting the bones are anatomically those of a non-calcifying plastic osteitis or periostitis; we find in the uncalcified subperiosteal tissue of rickets the interstices of the finely cancellous "bone" filled with a cellular connective tissue in which scanty numbers of lymphocytes and polymorphonuclear leucocytes occur.

Not only may rickets be excluded in Pollard's case, but so may syphilis. There was no history or evidence in the patient of congenital syphilis; and the new formation differs from a syphilitic one in its circumscription amidst the original bone. Its mere volume might be matched in a syphilitic infection. In the museum of St. Mary's Hospital there is the ulna of a syphilitic infant, which shows a pyriform enlargement involving the upper two-thirds, the swelling attaining a diameter of 2·5 cm., whilst the diameter of the unaffected portion below is but 0·5 cm., and the extreme length of the bone only 10 cm. The shaft within the periosteal node has been so rarefied as to be unrecognisable, though some indication of it is afforded by the vertical trend of the cancelli in the deeper part of the swelling; the medullary canal is not filled in; other of the bones were likewise affected.

Into the definition of a cancellous osteoma doubtless we should now insert, as a necessary part, the presence of marrow, *i.e.*, of adipose or of true medullary tissue, or both. But although such a basis of distinction between a true cancellous osteoma and an inflammatory formation might be made during the progressing stage, it must be allowed that the connective tissue filling the interstices of an inflammatory production of new bone might at a later date become infiltrated with fat and so acquire the characters of the unessential adipose element of medulla. In the case under discussion, however, the growth following the fracture was of recent date, and it can only be classified as a true cancellous osteoma.

In both Pollard's case and that described by Mr. Pitts and myself, the circumscription of the newly formed, imperfectly calcified, and finely cancellous tissue is striking; and I may add yet another illustration of what I believe to be the same anatomical condition. In the goat it is not rare to meet with an enlargement of the lower jaw, in which the bone is replaced by a finely porous osseous tissue, so soft that microscopic sections can be readily prepared without any decalcification. The disease has been described in the *Transactions of the Pathological Society*, xl., by Mr. W. G. Spencer. It is illustrated by many specimens in the Museum of the Royal College of Surgeons, and I have myself had the opportunity of examining others which were sent to me by Professor Sherrington when Superintendent of the Brown Institution—the same source, indeed, which furnished Mr. Spencer with his. It is observed in goats of from two months to three years of age, and chiefly in females kept for milking. The age of some of the animals excludes rickets, nor are the epiphysial signs of this disease present in the long bones in those examples where the animal is young, *e.g.*, two months.

In an early stage there is observed a symmetrical swelling of the lower jaw, between the angle and the molar teeth, the jaw feeling softer and being capable of indentation; as the disease progresses the whole of the jaw, the body, and rami become involved. The upper jaws may exhibit similar disease. The close connection of the morbid changes with the teeth is noteworthy; it suggests the origin of an infective

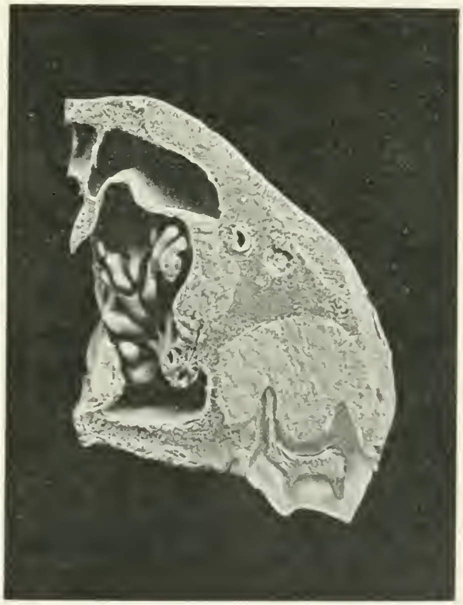

FIG. 8.

A section of the superior maxilla of a goat, showing a somewhat circumscribed interstitial formation of imperfectly calcified tissue which has grown within and replaced the original bone. The new formation immediately surrounds the fangs of one of the molar teeth. Natural size. (715) D, Museum Royal College of Surgeons.)

Maxillae superioris caprae sectio. Telae connexivae osteogeneticae imperfecte calcificatae monstratur moles quae pro telâ normali substituitur et dentis radices amplectitur. Magnitudinis naturalis.

process occurring by way of the alveoli. In some cases the ends of the long bones present a similar replacement of normal by abnormal tissue. What appears to be the same disease, says Mr. Spencer, had been previously noted by many veterinary surgeons in the horse. My own sections, made without any artificial decalcification from one of these diseased jaws, show a finely cancellous bone, some of the trabeculæ of which are quite devoid of earthy salts, yet they otherwise present all the detailed structure of normal bone and are closely surrounded with osteoblasts. In other trabeculæ central calcification has occurred. The material within the cancellous spaces is throughout a cellular connective tissue, without proper medullary cells or fat; a few osteoclasts are present. The connective tissue is of a looser or more open texture than in Pollard's specimen or in the tibia described by myself. Their circumscription would be the only ground for classing these different formations amongst tumours. In such a classification they would be interstitial osteomata, *i.e.*, tumours growing interstitially in, and replacing, the bone, as differentiated from exostoses or enostoses; and to indicate their imperfect classification they would be "osteoid osteomata." The absence of medullary tissue, however. forbids the adoption of such a nomenclature.

The new formation described in the present communication is, on the contrary, a true osteoma, the growth of which followed upon a fracture, for not only is it non-invasive, and therefore not sarcomatous, but it has throughout the gross structure of cancellous bone, the spaces of which are everywhere filled with fat, between the cells of which there lie strands of true medullary tissue comprising finely granular and coarsely granular myelocytes, with lymphocytes, intermingled with red corpuscles. The subsequent growth of a subperiosteal osseous tumour on the opposite right femur of the same child, and which, so far as can be judged by the skiagram, is of the same kind, is remarkable, and still more so is the appearance of a further fusiform osteoma around the middle of the shaft of the right tibia, and of less considerable, flattened and subdivided outgrowths from the radius and ulna. The formation of one of these, viz., that on the tibia, followed an injury—a fall on the pavement. The amount of bone formation and its multiplicity recall the cases of multiple osteoma which led Virchow to retain with some approval the traditional term "ossific diathesis," meaning by this an idiosyncrasy for producing bone under conditions which do not avail in normal individuals. In myositis ossificans the same thing is witnessed. It is impossible to explain the widespread formations of bone in this disease without assuming some general predisposition. The mere fact that the muscles are first inflamed does not of itself suffice, for myositis does not in other cases terminate in such a way. There is a myositis fibrosa as well as a myositis ossificans. The osteomata springing from the radius and ulna are clearly, as shown by their flattened form and their position, of the kind which grow into muscular attachments, aponeuroses, and ligaments. We may find the same combination of such ill-defined osseous outgrowths and true osteomata in other cases of multiple exostosis.

In the Museum of St. Thomas's Hospital there is the skeleton of an adult affected with multiple bony tumours (Spec. No. 602). Some of these are pedunculated and have a lobulated surface, but in certain situations the excrescences take the form of exaggerated muscular and fascial processes.

Some may be inclined to place the present case amongst those of "redundant callus" formation. But such a clinical group is too ill-defined. It comprises examples of different pathological conditions, and it is more accurate to differentiate such, as far as is possible, according to their anatomy and pathogenesis. Certainly, in the majority of cases, the formation of redundant callus after fracture results from want of immobility during the repair. This of itself may be sufficient, from the mechanical irritation it involves, and explains why such examples are chiefly furnished by fractures of the upper end of the femur.

But here, again, the influence of collateral conditions must not be overlooked. In some cases of tabetic arthropathy the amount of new bone formation accompanying the destruction of the articular ends of the bones is very striking. And in the repair of a fracture in tabetic patients, as Mr. Battle has pointed out, huge formations of bony callus may take place.[1] The exact pathology of such results cannot be given. If the excessive formation of callus is to be referred to an altered trophic influence resulting from the disease of the central nervous system, it must be remembered that this *may* act by favouring the action of other factors, as well as in the more hypothetical manner suggested by Mr. Battle in the earlier part of this communication. The possibility of an auto-bacterial infection of the injured parts leading to a chronic inflammation must be kept in mind, *i.e.*, an inflammation arising from the action of micro-organisms already in the tissues, the action of which was held in check until the advent of the injury. And, apart from tabes, there is no doubt that a proper inflammatory process must be invoked to explain the exuberant formation of callus in certain cases.

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[1] *Lancet*, 1904, ii., p. 580.

In regard to the transport of living bacteria to the organs of healthy animals during life, I may cite some remarks from a communication of my own in the *Transactions of the Pathological Society*, (1902, liii., p. 436): "From the early experiments of Tiegel[1] up to the latest of Ford, the solution of this question, presenting at the outset so delusive an exterior of simplicity, forms quite an episode in bacteriological history. The presence of bacteria in a living state, but one, so to say, of suspended animation, is a possibility not foreseen in the early stages of the inquiry. The experiments of Tiegel (who concluded that the normal viscera are *not* free of living bacteria) were made by submerging pieces of the freshly excised organs of animals in molten paraffin (110° C.—150° C.), which was then allowed to set. After a variable time of from four to twelve days the paraffin block was broken and the contents examined microscopically.

The experiments of Ford[2] consisted in transferring portions of the organs of healthy animals, suddenly killed, into nutrient media, the surfaces of the pieces themselves being first sterilised by cooking in the flame. Inoculations were made at the end of the first and third days by drawing up some of the juice of the organ in a sterile glass pipette, subsequent inoculations being made until positive results were obtained or a sufficient time had elapsed to show that the viscera were absolutely sterile. The cultures arose from those inoculations which were made from the original material on the sixth, seventh, or eighth day, or even as late as the seventeenth. The result was that in healthy domestic animals 70 per cent. showed bacteria in the internal organs provided that a sufficient time was allowed to elapse between the removal of the organs and their final examination. From the guinea-pig, for example, were isolated *Staphylococcus pyogenes aureus* and *albus*, *Bacillus proteus*, *B. coli*, *B. subtilis*, *B. mesentericus*. The tubercle bacillus is not given in the lists. The observations of Pizzini are therefore worth bearing in mind. Pizzini[3] inoculated guinea-pigs with the lymphatic glands from the bodies of forty persons who had died of non-tubercular affections; tuberculosis resulted in 42 per cent. of the experiments, the bronchial glands being those which most frequently gave a positive result. In the human subject the results, so far as the peritoneum is concerned, have been confirmed by Mr. L. S. Dudgeon, who has cultivated the *Staphylococcus albus* from the blood effused into the peritoneum in 17 cases of extra-uterine pregnancy, 2 of rupture of the spleen, and 1 of rupture of the liver.[1] In the guinea-pig Mr. Dudgeon also finds the *Staphylococcus albus* often present as a normal inhabitant of the omentum, and he points out that the presence of non-bacterial substances injected into the peritoneal cavity (chalk suspended in salt solution) appears to increase its activity there in the majority of cases.[2]

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[1] *Virch. Archiv.*, 1874, lx., p. 453.

[2] *Journ. Hygiene*, 1901, i., p. 277.

[3] *Zeitsch. für klin. Med.*, Berl., 1892, xxi., p. 329.

Malgaigne[3] cites at length the case of a man who sustained a fracture of the upper third of the right femur from a fall from a horse. The case was treated by Dupuytren by means of direct extension, and afterwards by the double inclined plane. This change of position caused the fragments to incline one over the other, marked swelling and great pain following. Callus formed slowly and remained deformed. The patient, nevertheless, got up. The callus swelled and became the seat of acute pain; the limb became œdematous and of violet colour. All the means of treatment adopted were unavailing.

Only towards the third year, under sea bathing, did the pain, swelling, and congestion in great part disappear. The two fragments were surrounded with a large mass of callus. Blisters, and finally an issue, were used. Some months later the limb recovered its colour and form. Malgaigne refers briefly to two other similar but less marked examples, and concludes by observing that this affection of the callus has the character of a chronic inflammation involving the parts. Here the view of an auto-bacterial infection best explains the result, the inflammatory process having, of course, stopped short of suppuration. This view is rendered still more probable by the case afterwards cited by the same author, where a man, aged 18, sustained a fracture of the middle of the femur, and attempted to walk and resume his work at the end of the fourth week. Six weeks later the limb had shortened 2 in., and the callus had acquired an enormous volume; abscesses had formed in many parts.

Another matter of surprise in connection with the present case is the spontaneous disappearance of the osteoma on the right femur. As shown by the skiagram taken in 1903, the lower half of the shaft of the right femur was then surrounded by a somewhat irregularly fusiform bony tumour, whilst the skiagram taken in 1907 showed that this had disappeared. Abernethy, nevertheless, has recorded what in this regard is a comparable case.[1] And it is noteworthy that in this, also, the appearance of the bony formations would follow the receipt of an injury. "A youth, aged about 14, was brought to me whose back was greatly deformed by irregular hillocks of earthy matter heaped up upon the spinous processes of the vertebræ. The ligamentum nuchæ was ossified, so that his head was immovably fixed, being drawn backwards and slightly inclined to one side. There were exostoses on the os brachii of both arms, and the tendinous margins of the axillæ were converted into bone, and pinioned his arms so closely to his sides that it was difficult to insinuate the fold of a napkin between them and his chest. There was an exostosis on the pelvis, between the sacrum and os innominatum, *and various others had formed at different times and disappeared*, but those which I have mentioned were permanent. Being a robust and spirited youth, he was disposed to exertion, and if, in a forcible effort to accomplish any purpose, which his manacled situation obliged him often to make, *he accidentally struck his head, or any projection of a bone, a temporary deposition of earthy substance in the injured part was always the result.* He had had the toothache a little before I saw him, and the remains of an exostosis, which had been considerable, still appeared on the lower jaw. Two years after the time I first saw him the youth came to London again, and the exostoses which I have described seemed to be pretty much in the same state; several new ones, however, had formed on the extremities; one in particular extended itself from the pelvis along the thigh in the direction of the sartorius muscle, and impeded the motions of the limb."

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[1] "The Bacteriology of Peritonitis," Dudgeon and Sargent, 1905.

[2] *Trans. Path. Soc.*, Lond., 1906, lvii., p. 171. L. S. Dudgeon and A. Ross.

[3] "Traité des Fractures et des Luxations," i., 1847

As already stated by Mr. Battle, the right lower limb of the boy's mother, a woman aged 24, was likewise amputated for what was clinically supposed to be a sarcoma, the operation having been carried out by Mr. Cotman, of Rochester, about a year after the limb of the child had been removed by Mr. Battle. The amputated limb, which had been buried since February 17, 1903 (the date of the operation), was exhumed in April and sent to me for examination, notwithstanding which, microscopic preparations of the tumour show quite clearly its histological structure, a preservation possibly in part due to the saturation of the tissues with the anæsthetic during the operation. The growth on the femur, after removal of the surrounding soft parts, presents itself as a bilobed or transversely constricted mass, which does not completely surround the shaft, the outline of which is traceable on one of its aspects, though misshapen from the growth of a long lamina of new bone, which is scarcely anywhere connected with the tumour, but resembles the expansions met with as intramuscular or intrafascial osseous outgrowths. In cross section the lesser of the two lobes or subdivisions of the mass measures 2½ in., and consists of a somewhat coarsely but fully hardened cancellous tissue, the spaces of which are filled with fat; the surface is closed with a thin layer of compact tissue. The larger lobe, which has a diameter in cross section of 4 in., presents a closer or finer cancellous texture, and is in places so imperfectly calcified as to be resilient, like sponge. The outer surface of this portion is lowly lobulated or tuberose, but intact and sharply defined; in places it is finely porous, elsewhere closed with a thin compact lamina. Portions of the outer surface are so imperfectly calcified as to be quite elastic. On longitudinal section the two lobes are found to be discontinuous, being separated by a sheet of fibrous tissue which reaches down to the remains of the shaft, one apposed surface being regularly convex and the other concave. The remains of the shaft, incorporated with the tumour, together with the medullary canal, which preserves its patency, are traceable in the section. In some situations the compact wall of the shaft is quite intact beneath the growth, the periosteum intervening, showing that a portion of the mass has grown around or overlapped the original bone.

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[1] Abernethy, "Lectures on the Theory and Practice of Surgery," 1830, p. 169.

Histology.—Sections of one of the low nodules of the superficial part of the larger of the two divisions of the growth were cut vertically to the free surface, the spot selected being one in which the tissue was resilient and sponge-like from imperfect calcification. The sections exhibit throughout a cancellous structure, with a uniform mesh, the trabeculæ composing which are somewhat coarse and are composed of hyaline cartilage. The spaces of this cancellous cartilage, as it might be called, are filled with a loose connective tissue, by no means very cellular, and neither the cells nor the fibrils of which have any definite disposition. From this inter-trabecular tissue the development of the cartilage is readily traceable. Cells lie against the trabeculæ in all stages of inclusion in a hyaline matrix; these cells, although somewhat small, are contained from the first in spherical spaces, and pass by all gradations into the larger spherical cells occupying the more central parts of the trabeculæ. Sometimes amongst the smaller cells in process of inclusion, or just included, characteristic groups of two occur with flattened sides of apposition; and nowhere do the enclosed cells present the flattened character of the proper osseous corpuscles in the lacunæ of membrane bone. If any true membrane bone is anywhere in process of formation, its amount is insignificant. In the more central parts of the trabeculæ the matrix is in different degrees calcifying, the cells themselves retaining their large size and spherical forms. The actual surface is composed of hyaline cartilage in which no calcification has taken place, the matrix being clear and quite devoid of granules.

The zone of cartilage bounding the surface is traversed with connective tissue so as to present a trabecular structure; but the spaces are narrower than elsewhere, and the connective tissue occupying them is more cellular. Both the trabeculæ (the cells of which are here smaller) and the tissue between them, however, merge directly, on the deeper aspect, into the larger-celled cartilage and into the loose connective tissue filling its cancelli, respectively. The tissue of the smaller spaces presents no sarcomatous characters; it is simply connective and chondrific. Sections of the deeper, somewhat harder, portions of the larger division of the growth, cut after decalcification in picric acid, display the same structure. The tissue in the cancellous spaces of the cartilage has none of the characters seen in a chondrifying sarcoma, but consists of loose connective tissue with cells devoid of any regular disposition and by no means conspicuous in numbers.

This tumour cannot be classed with inflammatory formations for the obvious reason that it is so extensively constructed of true cartilage. The ultimate transformation of large areas of it into cancellous bone, holding fat, removes it from the group of sarcomata. The new growth finds its place amongst those described and named by Virchow osteoid chondromata. There is no reason for regarding it as a malignant formation, nor does Virchow's name either exclude or imply malignancy. We may relate the neoplasm in its general characters to the peripheral chondromata which grow around the larger bones in the adult, and in which extensive formation of true cancellous tissue may take place—ossifying chondroma. In both forms the bulk of the growth is cartilage, but in the ossifying chondroma the formation of bone proceeds after the normal type; the matrix of the cartilage is first calcified, and the calcified tissue with included cells is then penetrated by vascular connective tissue, from which is laid down membrane bone in the spaces resulting from its destruction. In the osteoid chondroma the formation is essentially cancellous. It might almost be said to consist of cancellous cartilage. The cancelli are formed of cartilage, and although secondary additions to the trabeculæ may be made from the connective tissue filling the cavities, this occurs without the original cartilaginous substance being removed. In the normal process of intracartilaginous ossification, the only remnants of the cartilage, it will be remembered, which form any constituent of the proper bone are the lines of calcified matrix between the rows of cartilage cells, the latter cells disappearing before the invading connective tissue. In the osteoid chondroma the tissue, indeed, recalls that produced at the epiphysial line in rickets, for in this disease the proliferating cartilage is riddled with the vascular ossific connective tissue from which membrane bone is formed, but without the cell-containing trabeculæ of the cartilage being removed.

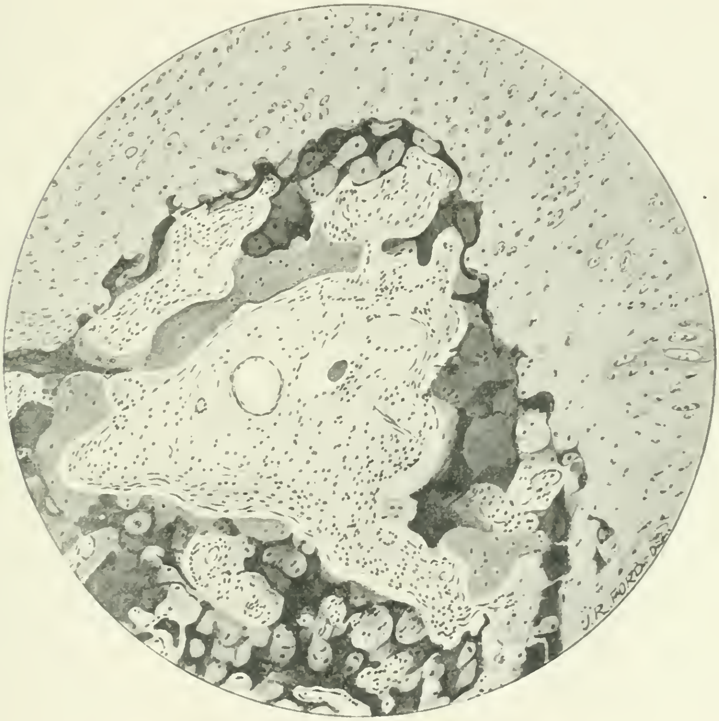

FIG. 9.

Portion of a chondroma, showing the manner in which true bone is produced, viz., by the removal of calcified cartilage and the substitution of membrane bone formed from the invading connective tissue. The calcified cartilage is distinguishable by the darker way in which it has stained. $\frac{2}{3}$ objective.

The conclusion at which we arrive in regard to this tumour is that it is benign, and that it is classifiable as an osteoid chondroma. The growth of the osteomata in the patient's two children becomes, thus, an example of the heredity of benign tumours, which is, in the case of chondromata and osteomata, so striking and well established.

ADDENDUM.

A younger brother of the patient was admitted, under the care of Mr. Battle, into St. Thomas's Hospital on February 3, 1908, for an extensive tumour of the right thigh. The patient, who was aged 3½, was sent up by Mr. Pitcairn, of the Rochester Hospital, where he had been admitted on July 11, 1907, for a simple fracture of the femur due to a fall. About three weeks after admission it was noticed that the thigh had become much larger than it should have been, and by December it measured 19 in. in circumference. Since that time there had been some diminution, the circumference of the limb being 14½ in. A microscopical examination of a small piece of the tumour removed by operation at Rochester was made by the Clinical Research Association, the report being as follows: "The specimen consists at one end of well-formed bone, at the other of young connective tissue, and in the middle shows newly formed cartilage in process of ossification." Under the X-rays the tumour exhibited an osseous structure exactly resembling that of the growth around the fractured femur recorded in the preceding communication.

The patient was shown at the meeting of the Medical Society of London, February 10, 1908; and excepting for the very large tumour, which was hard throughout and without pain or tenderness, he appeared in fair health. An account of this case, with an accompanying photograph, will be found in the *Transactions of the Medical Society*.

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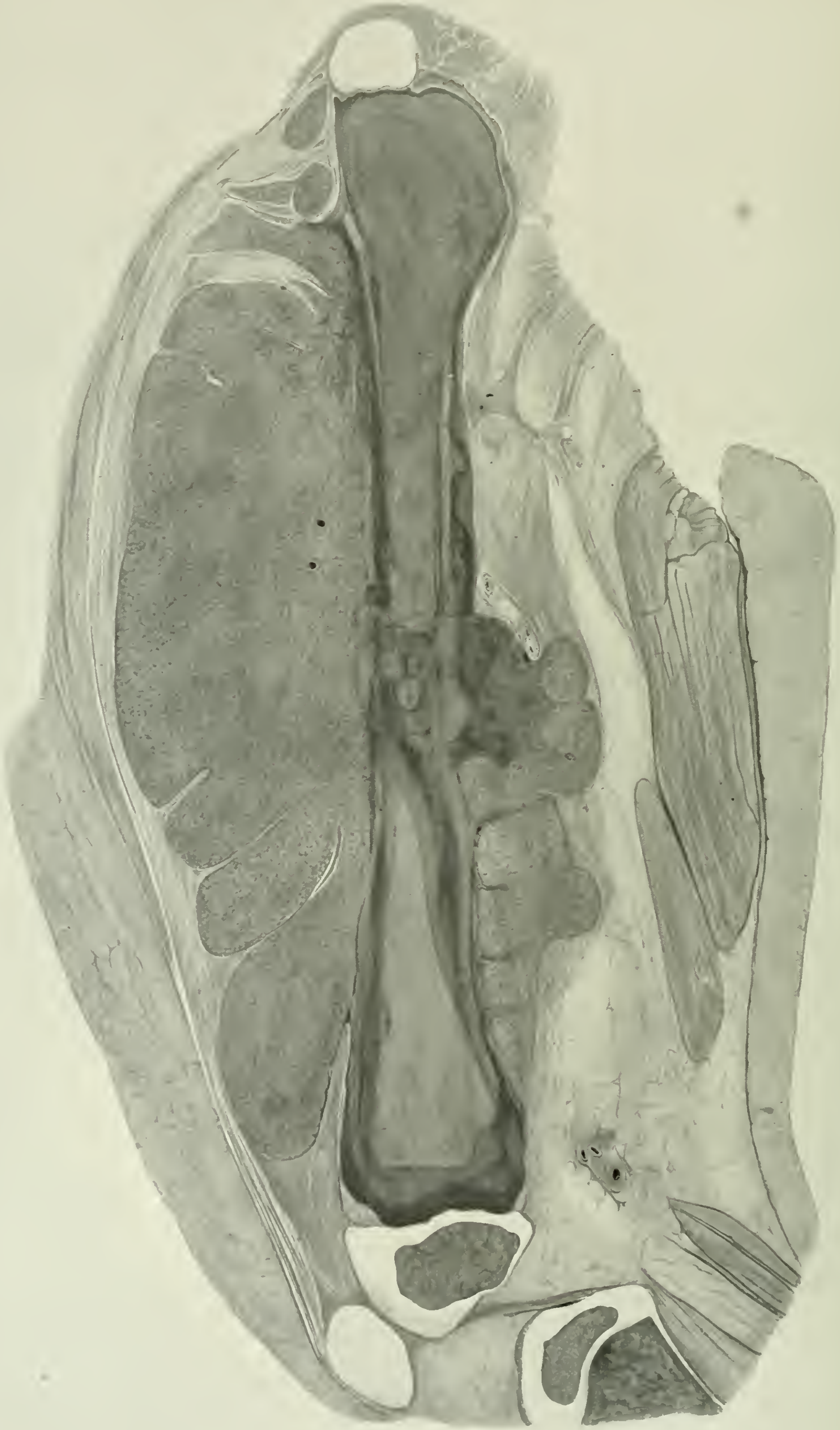

J.R. FORD DEL

EXPLANATION OF PLATE.

Illustrating the communication on Diffuse Osteoma of the Femur following Fracture.

By W. H. Battle and S. G. Shattock.

A sagittal section of the thigh, showing the extensive growth of normal finely cancellous tissue around the shaft of the femur, which followed a fracture of the bone. The fracture has healed without displacement. The shaft is traceable through the tumour, no extension of which occupies the medullary canal.

Slightly reduced. (Mus. Royal College of Surgeons.)

Tabula.

Ad dissertationem "De osteomate diffuso post fracturam evoluto" illustrandam.

W. H. Battle et S. G. Shattock.

Crus in longitudinem sectum. Neoplasma monstratur praegrande, ex telâ osseâ constans, quod sicut sarcoma, femur amplectitur.

Fractura sine fragmentorum dislocatione reparata est.

In medio tumore discernitur femur cujus neque paries compactus nec cavitas medullaris invaditur.

Figura aliquantulum diminuitur.

Tuberculous Endocarditis.

By W. O. MEEK.

THE occurrence of recent endocardial lesions in cases of infection with the tubercle bacillus has been observed on numerous occasions. In very few, however, of the recorded cases has an undoubted connection between this bacillus and the endocarditis been proved to exist. In certain text-books of medicine the tubercle bacillus is mentioned as a possible cause of endocarditis, while other authors state that, though it may cause tubercular nodules on the cardiac valves, it does not produce true vegetative or ulcerative lesions. The following two cases were examined recently in the Pathological Department of St. Thomas's Hospital. One, at least, appears to be a true example of a vegetative endocarditis due to the tubercle bacillus.

CASE I.

Clinical Account.—A. C., a male, aged 16, was admitted to hospital on August 28, 1907, under the care of Dr. Sharkey. There was a strong family history of pulmonary tuberculosis on the paternal side, the patient's father having died of that disease. No history of previous illnesses nor of any rheumatic manifestation was obtained. In the summer of 1906 the patient had slight hæmoptysis with cough. The cough persisted during the ensuing winter. He was admitted with a recurrence of hæmoptysis which had lasted for four days, but which ceased soon after admission. Signs of tubercular disease were found in the lungs and there was irregular pyrexia. Open air treatment was adopted. By the end of September the general condition showed considerable improvement and the fever was less. On September 30, however, the patient had a rigor with rise of temperature to 105·8° F. This was repeated the following day, and from this time till death there was continuous pyrexia. Generalised bronchitis ensued, together with enlargement and tenderness of the spleen; the patient's condition gradually became worse and he died on October 22. During the whole of his stay in hospital a murmur was audible over the cardiac area. The characters were those of a hæmic bruit.

Post-mortem Examination.—This was performed on October 23. A condition of well-marked generalised miliary tuberculosis was found. The upper lobe of the *right lung* contained a cavity the size of a walnut, lined by caseous material and communicating with a medium-sized branch of the pulmonary vein. In the surrounding lung tissue were a few small caseous areas. Situated immediately under the pleura, and in the neighbourhood of the cavity, were two pale, solid, wedge-shaped areas which, on microscopical examination, were found to be anæmic infarcts. No naked-eye evidences of disease were seen in the main pulmonary vessels. The *heart* showed slight general enlargement and weighed 12 oz. The cardiac muscle was pale and soft. On the ventricular surface of the aortic, and on the auricular surface of the mitral valve segments were a row of recent small vegetations. They were regularly disposed on each of the cusps and were situated a short distance from the free edges of the segments. In the case of the aortic valve the vegetations were exceedingly minute and white in colour. On the

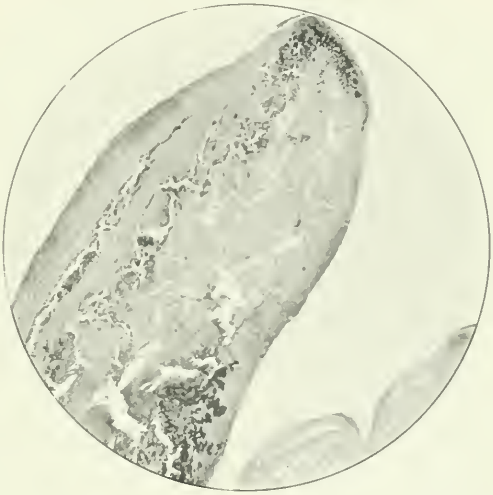

FIG. 1.

A low power view of the distal portion of the vegetation from Case I. This portion of the nodule consists of granular necrotic material containing very numerous dark masses of tubercle bacilli.

Zeiss objective A. No. 2 eyepiece. Stained with carbol-fuchsin and hæmalum.

mitral cusps they were somewhat larger, the largest being the size of a No. 5 shot, and were light brown in colour. In both situations they were friable and soft. There was no evident destruction of valve tissue and no signs of pre-existing valvular disease. There was no increased vascularity around the bases of the vegetations. The valves on the right side of the heart were healthy. The *spleen* was much enlarged (16 oz.), firm and dark. It contained numerous tubercles with areas of necrosis and several wedge-shaped recent infarcts. The *liver* also contained a small infarct at its free edge.

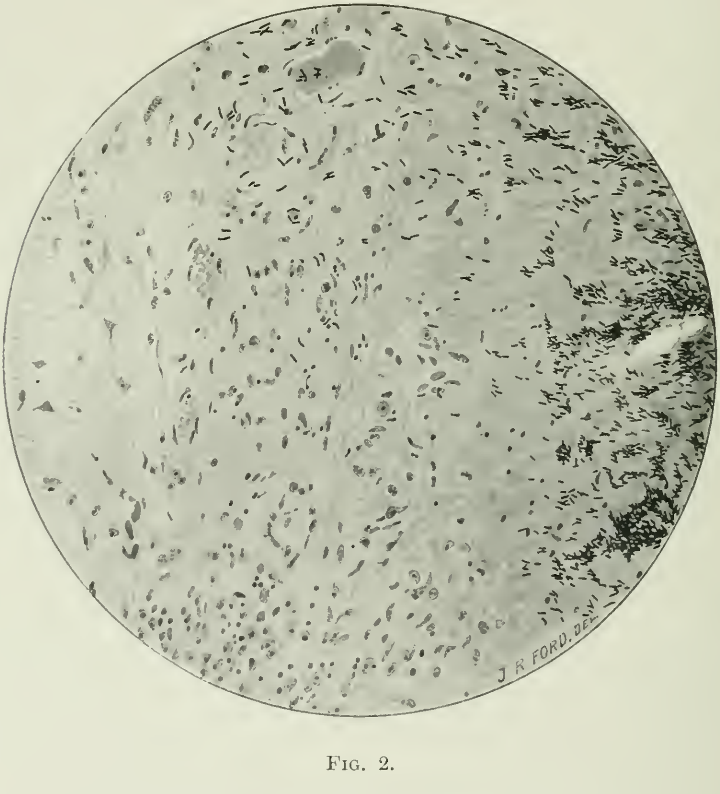

Fig. 2.

A portion of the base of the vegetation from Case I., showing the zone of cellular infiltration at the junction of the vegetation and valve. Bacilli are scattered throughout this area. In the upper part of the figure is a small necrotic focus with included bacilli.

N.B.—The size of the individual bacilli is somewhat exaggerated.

From a drawing. Zeiss objective D, No. 1 eyepiece. Stained with carbol-fuchsin and hæmalum.

Histological and Bacteriological Examination.—Microscopical examination of sections taken through the centre of one of the vegetations from the mitral valve shows it to consist in its distal portion of a granular, lamellated, necrotic material staining a deep pink with eosin. Throughout this portion are irregular clefts or spaces, some containing masses of red blood-cells and a few leucocytes. At the base of the vegetation and at its junction with the valve is a zone of cellular infiltration. The cells present consist of various types of mononuclear cells, including a few plasma cells and numerous spindle-shaped elements. Nuclear fragmentation can also be seen and a few small areas of necrosis or caseation. In one place there is a circular caseous area showing a central clump of bacilli. The spindle-shaped connective tissue cells are extending into the necrotic portion of the vegetation. A few blood-vessels are seen among the cells at the base. There is no evidence of

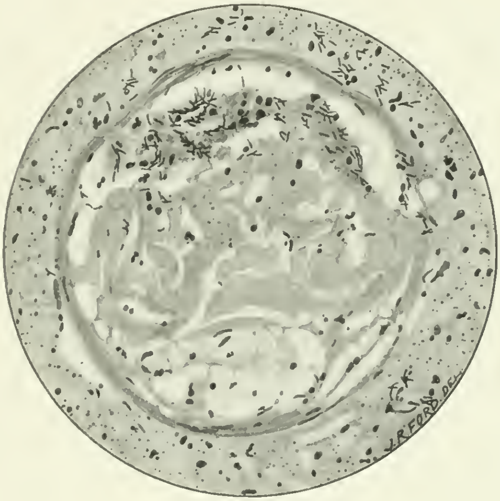

FIG. 3.

Drawing of a small vessel in the splenic pulp of Case I. The vessel is occluded. The greater part of the lumen is occupied by thrombus. The upper portion of the lumen shows the presence of many bacilli, surrounded by necrotic material, differing somewhat in structure from the subjacent thrombus, and suggesting an embolic origin. The surrounding zone of splenic tissue is in a condition of necrosis.

Zeiss objective D, No. 1 eyepiece. Stained with carbol-fuchsin and hæmalum.

giant-cell formation nor any signs of an acute inflammatory condition. Sections through the edge of the vegetation show that, in this situation, the zone of cellular infiltration is wider, but on the whole evidences of inflammatory reaction are not marked. Sections stained with orcein and methylene blue show that the vegetation is situated above the elastica. Other sections, stained with carbol-fuchsin, decolorised with nitric acid and counter-stained with hæmalum, show the presence of enormous numbers of tubercle bacilli throughout the vegetation. Not only are they present in the superficial part of the nodule but they extend right down to its base and are lying thickly scattered among the cells at the junction of vegetation and valve. In places they form large, densely staining masses or clumps such as are commonly seen in avian tuberculosis. Various staining methods for the detection of other micro-organisms were employed but none were found. Microscopical examination of an infarcted area of the spleen shows extensive necrosis, with thrombosis of small vessels. In some of these vessels emboli, containing numerous tubercle bacilli, can be seen. The other solid viscera examined all showed well-marked microscopical evidence of miliary tuberculosis, a noticeable feature being the great number of small areas of necrosis. Post-mortem cultures from the splenic pulp in broth and on agar were sterile at the end of four days.

Case II.

Clinical Account.—G. G., male, aged 16, was admitted to St. Thomas's Hospital on September 30, 1907, under the care of Dr. Hawkins. There was a family history of tuberculosis. There was a history of measles in infancy, but otherwise the patient had always been healthy and strong, and had never suffered from rheumatism. The illness for which he was admitted was of three weeks duration, the onset being acute, with abdominal pain, vomiting and constipation. A tender mass was felt in the right iliac fossa and the temperature was raised. There was no leucocytosis. Laparotomy was performed and the condition found to be a tuberculous peritonitis. The wound healed by first intention, and the general condition remained stationary till October 17, when signs developed in the lungs. He went rapidly downhill, and died on November 1. At no time were signs noted pointing to any cardiac valvular lesion.

Post-mortem Examination.—This was performed by Dr. Box on November 1. Generalised miliary tuberculosis was found, with extensive intra-abdominal tubercular disease, the oldest lesions being situated round the cæcum. The heart and pericardium were healthy externally. A fringe of rather exuberant granulations was discovered on the auricular aspect of the free edges of the mitral valve.

Histological Examination.—On microscopical examination of one of the vegetations it is seen to consist of necrotic material, similar to that seen in the distal portion of the nodule in the previous case. This necrotic tissue shows many crevices in which are swollen mononuclear cells, resembling endothelial cells, and some of them contain vacuoles in their cytoplasm. At the junction of vegetation and valve is a zone of proliferated mononucleated cells, but the exudation of cells is only slight. Tubercle bacilli are seen throughout the vegetation, but not in large numbers. Nowhere is the typical histological picture of tuberculosis visible. In the blood which is adhering to the edges of the vegetation tubercle bacilli can be seen in sections stained by various methods.

The proof that a micro-organism, found in a lesion on a cardiac valve, is itself the cause of such lesion, is often a matter of great difficulty. More especially is this difficulty present in the case of the tubercle bacillus, with which we are accustomed to associate a definite histological picture.

Marshall [1] states that to furnish such proof in the case of the tubercle bacillus the following microscopical appearances must be observed : "The bacilli must be demonstrated, not on the surface, but in the depth of the vegetation, where their presence could not be due to accidental contamination." "It must be shown by staining sections that the bacilli have not simply lodged in the vegetation, but are actually associated with the lesions typical of tuberculosis, and finally, as Benda emphasises, the elastic tissue stain must be employed to prove that the process is above the elastica, that is, in the endocardium, and not below, that is, in the myocardium."

Sorgo and Suez [2] in a recent article on this subject accept only nine cases as showing an undoubted connection between the tubercle bacilli and the endocardial vegetations in which they were found. They refuse to admit any case which does not present some histological appearances typical of tuberculosis. In only one [3] of their nine accepted cases, however, were giant-cells mentioned as found in the lesion ; in the remainder, microscopic areas of caseation were present.

The first of the two cases reported to-day satisfies all the above mentioned requirements, with the possible exception that it does not show the presence of giant-cell formation. But it must be remembered that tubercle bacilli *may* be present in tubercular lesions without any giant-cell formation, so that their presence is not an essential of the tubercular process. Small necrotic or caseous foci are, however, to be seen in the deeper parts of the vegetation, and in some of these foci bacilli are visible.

In explanation of the slight amount of cellular exudate at the junction of valve and vegetation, it is an important fact that marked inflammatory reaction is not an invariable feature in the formation of infective cardiac vegetations. To furnish a site for their formation it is only necessary that the infecting micro-organism shall lodge upon the surface of the valve and produce necrosis of the intima, the accompanying inflammatory reaction being, perhaps, extremely slight. This absence of marked inflammation is, indeed, often noticeable in lesions of the cardiac valves arising in the course of general infections by the pyogenic cocci. In the present case, sections of other organs show a large number of small areas of necrosis, sometimes with a central clump of bacilli, and round these areas there is, in many cases, neither giant-cell formation nor marked cellular proliferation.

The occurrence of rigors during the patient's illness, together with the post-mortem findings of cardiac vegetations and visceral infarctions, suggested, at first sight, a mixed infection. Histological and bacteriological examinations, however, have failed to demonstrate any other organism. The whole picture of the case is that of a bacteræmia due to the tubercle bacillus, with multiple emboli.

Braillon and Jousset [4] have reported a case showing the clinical signs of septicæmia. Blood drawn from a vein during life contained tubercle bacilli. At the post-mortem examination vegetative endocarditis was found without any appearances of pulmonary or lymphatic tuberculosis. Tubercle bacilli were present in the vegetations. This case they regard as one of primary tubercular endocarditis.

With regard to the second of the two cases reported to-day, although tubercle bacilli are present in moderate numbers in some parts of the vegetation and in the adherent blood, they are seen only very sparingly in the deeper layers; moreover, the histological picture shows no lesion typical of tuberculosis. This case, then, does not fulfil the required conditions, and must be classed with other reported cases in which the connection between the vegetation and the bacilli found in it is not definitely proven.

In conclusion I wish to thank Mr. Shattock and Mr. Dudgeon for their kind advice and help, and Dr. Sharkey and Dr. Hawkins for permission to make use of the material obtained from these cases.

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Sarcoma of the Rectum in a Boy, aged 10.

By Cecil Rowntree.

The case is that of a boy, aged 10, who, in July, 1906, was admitted to the Middlesex Hospital for chronic intestinal obstruction, which had been present for three months. Within a week of admission symptoms became more acute, and Mr. Andrew Clark performed laparotomy. The operation disclosed the fact that the obstruction was due to the condition of the rectum, which was thickened, infiltrated and irregular and obviously the seat of a new growth. Colotomy was accordingly performed, and the boy left the hospital on August 30, free from acute symptoms. Very shortly afterwards, however, a swelling appeared on the forehead just above the right eye, and rapidly increased in size until it formed a hemispherical projection about 1 in. in diameter. At this stage peritonitis supervened, and the boy died on October 18, six months after the appearance of the first symptom.

I was asked to investigate the case by Dr. Richardson, who was in charge of the patient at the time, and below is an account of the appearances found post mortem:—

The body was extremely emaciated and showed a colotomy wound just below the umbilicus. On opening the abdomen general suppurative peritonitis was found to be present, and the rectum was imbedded in a large mass formed in part by new growth, partly by inflammatory tissue. When the bowel was split open it was seen that for an extent of about 4 in. the mucous membrane of the rectum was indurated, thickened and irregular, but there was no breach of surface except for a small ulcer on the anterior wall in the middle of this indurated area. The ulcer communicated by a short track through the cellular tissues with the peritoneal cavity, permitting the escape of intestinal contents and so accounting for the peritonitis.

Further examination showed that growth had extended from the rectum into the pelvic cellular tissues, invading the ureter in its course and forming a continuous mass with the pelvic lymphatic glands, which were much enlarged by rather waxy-looking whitish growth. The lumbar glands as far up as the pancreas were occupied by similar growth and formed large masses obscuring the aorta and vena cava. There was also deposit in a few of the mesenteric glands and in the mediastinal glands along the course of the internal mammary arteries. The viscera were free from metastatic growth, but there was a small deposit under the periosteum of the fifth rib on the right side; two masses of growth were found growing from the frontal bone, one of which had extended right through the bone, lifting up the pericranium in front and indenting the surface of the brain behind. Yet another deposit was found lying under the periosteum of the innominate bone on the right side. This last growth was very dense and appeared to contain a good deal of bone in its substance.

The microscopic characters of the primary growth and its various metastases vary considerably. A section through the wall of the rectum shows thickening of the submucous coat, which in places is covered by normal epithelium, while in other parts the epithelium is replaced by a delicate fibrous meshwork in which are scattered cells of irregular shape. These cells are arranged either singly or in small clumps of two or three and stain in a characteristic way with Pappenheim's stain, which clearly shows them breaking through the muscularis mucosa. In the submucous coat the growth forms large masses which show much degeneration, which is apparently mucoid in nature. The muscular coat has a very striking appearance. It is much thickened, and between the bundles of circular muscle fibres are long slender columns of swollen and degenerate cells, which give the staining reactions of mucin. At the outer limit of the muscular coat these columns of growth have more room for extension and form large irregular masses, which extend into the perirectal tissues.

The lumbar glands are occupied by growth which differs from the primary tumour by possessing a well-marked fibrous stroma, in the meshes of which are small masses of cells which again show such marked mucoid degeneration that it is difficult to be certain of their nature. The growth in the frontal bone is more cellular, the stroma is much less prominent, and degeneration of the cells is again a prominent feature.

In the deposit in the innominate bone the appearances are quite different; the fibrous stroma so well marked in the lumbar gland is here replaced by a well-formed bony skeleton, the spaces in which are filled with growth having all the appearances of mixed-cell sarcoma. Whether the bone in this deposit is part and parcel of the growth or merely the result of periosteal activity is not certain, but in view of the fact that none of the other deposits showed any bony tissue, I am inclined to take the view that the bone is the result of irritation of the periosteum and not a metaplasia of the cells of the growth.

The appearances in the last section leave no reasonable doubt that the case is one of mixed-cell sarcoma, which apparently originated in the mucous membrane of the rectum, although a possibility that occurs to one is that the case is in reality not a primary growth of the rectum at all, but that the growth on the innominate bone was primary, and that the rectum was invaded secondarily from the presacral glands. This view is negatived, however, by the condition in which the rectum was found at the time of the operation, by its microscopic appearances, and by the distribution of the secondary deposits.

All varieties of malignant disease of the rectum are rare in early life. Of 591 cases analysed in the Cancer Research Laboratory of the Middlesex Hospital, in only 6 were the patients aged under 30. Of these 5 were said to be carcinomata, 3 males, aged 17, 22, and 23, and 2 females, aged 20 and 29 respectively; and in 1 case, that of a boy, aged 16, the growth was described as a sarcoma. In this case there was a large mass of growth in the rectum, and when the abdomen was opened for the purpose of performing colotomy, it was found that the peritoneum was studded with small masses of growth which were said to have the appearance of grains of boiled sago. A small piece was removed for microscopic examination, and the section shows a portion of omentum infiltrated with degenerated new growth of sarcomatous, or possibly endotheliomatous, nature. It is certainly not a carcinoma.

Sarcoma of the rectum appears to be very rare at any age. Sir Charles Ball, in his book on diseases of the rectum, illustrates a case in an adult which has some points of similarity to the case under consideration. In his case the bowel, for a space of 4 in., was the seat of a diffuse sarcomatous infiltration over which the mucous membrane was thickened and irregular, but, as in my case, was not ulcerated. This absence of ulceration of the mucous membrane, in spite of the extensive growth in the underlying tissues, is rather a remarkable feature, and may be compared to the condition which obtains in the so-called "leather bottle" cancer of the stomach.

On the Action of Vesical Calculi upon a Photographic Plate in the Dark.

By HECTOR A. COLWELL.

IN 1906 Dr. Lazarus-Barlow published a paper in the "Archives of the Middlesex Hospital" dealing with the electrical and photographic phenomena exhibited by certain substances supposed to be etiologically associated with carcinoma. Among other substances thus experimented upon were some cholesterin gall-stones, which, when placed upon a photographic plate in the dark, produced very marked effects.

Dr. Russell, in some papers which he communicated to the Royal Society, had previously noted that portions of various woods similarly exposed gave such definite results that the grain of the wood was exactly reproduced.

At Dr. Barlow's request I undertook the examination of some urinary calculi derived from the bladder. These calculi were, with one exception (a predynastic Egyptian calculus from the Museum of the Royal College of Surgeons), specimens from the Museum of the Middlesex Hospital; they had been halved, and the newest was about forty years old—the oldest being one which was removed by Percival Pott (1713-1788).

In order to eliminate the possible effect of sunlight, as shown in Dr. Russell's work, these calculi were placed in the dark in separate boxes in a warm, dry room for six months before experiment was made with them. They were then taken to the dark room, where even the red light was extinguished while they were removed from their boxes, and dealt with as follows: The calculus, with its cut surface downwards, was placed upon the sensitised side of a photographic plate ("Imperial Special Rapid"), and, with the object of cutting out any external influences which might act upon either calculus or film, the calculus and plate were wrapped in successive sheets of (1) thin filter paper, (2) a sheet of filter paper saturated with paraffin wax, (3) a piece of black "needle paper," and (4) the whole placed in a tin box. The tin boxes with their contents were then placed in a light-tight wooden box, removed to an incubator, and kept at 53° C. for ninety-six hours. After this they were again removed to the dark room, and developed practically in the dark, the red light being only used for two purposes: (1) when the calculus was removed from the plate to determine whether any dust or particles of the stone had adhered to the plate, and (2) at intervals to watch the progress of development. Eighteen calculi were examined by this method, of which thirteen produced definite effects and five made no apparent change in the film upon which they were placed. In the 13 cases producing a positive result a definite shadow was produced, in many cases pale, but, where present, of sufficiently definite outline for the portion of the calculus which produced it to be determined. This is in marked contrast to the results obtained by Dr. Lazarus-Barlow in his experiments with cholesterin gall-stones, where the effect was visible as a local dense blackening of the plate with no differentiation of structure.

In view of the fact that certain radiations from recognised radioactive substances traverse a sheet of mica, experiments with the calculi were conducted under these conditions, but in no case with a positive result.

The following notes show the results obtained from nine of the calculi which gave a positive result:—

No. I. (calculus 64).[1]—A calculus of laminated structure composed of uric acid, removed from a man, aged 66. The outer compact layer produces no effect, but a darker and less compact layer shows a well-marked elliptical shadow about 0·25 cm. in thickness; internal to this are the faint marks of certain other laminæ. The central compact nucleus gives no effect.

No. II. (calculus 65).—A calculus of ammonium urate with some phosphates (Ca, Mg) shows a central more compact nucleus, which is succeeded by more porous portions, which are most porous at the periphery. The nucleus gives no effect, and therefore appears as a clear space; this is succeeded by a dark band, which in turn is succeeded by a clear band, and this latter by a more or less punctate shadow, which, however, at the margins preserves the outlines of the original section.

No. III. (calculus 69).—The calculus is more or less pear-shaped, yellow in colour, and contains an oval whitish nucleus at the smaller extremity. This latter nucleus appears to consist largely of ammonium urate and the rest of the specimen of uric acid. The broader portion shows a punctate shadow, the ammonium urate portion no effect.

No. IV. (calculus 70).—An oval calculus composed of uric acid. It shows a compact nucleus which gives no shadow, the surrounding more porous material produces a punctate shadow.

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[1] These numbers refer to the Museum Catalogue of the Middlesex Hospital.

No. V. (*calculus* 76).—This is described as a calculus composed of uric acid with a little ammonium urate; it was removed by Percival Pott, and is therefore at least 120 years old. The photographic plate shows a fairly well-marked outline, which corresponds with a more or less compact external layer in the calculus itself. The more internal portions show varied and irregular markings, and the most compact portion at the centre of the calculus gives no shadow.

No. VI. (*calculus* 78).—This is mainly composed of ammonium urate and has a compact nucleus, surrounded by more porous layers and bounded by a compact white layer. The layer immediately surrounding the nucleus gives a well-marked dark shadow, and the junction of the outermost compact layer with the middle portion a distinct band.

No. VII. (*calculus* 85).—A calculus with a nucleus consisting of ammonium urate, which is surrounded by mixed phosphates mixed with urates. The central compact nucleus gives no shadow; the surrounding parts two well-marked bands, separated by a clear space.

No. VIII. (*calculus* 94).—This calculus consists mainly of calcium oxalate surrounding a compact mass of uric acid. The nucleus produced no effect upon the photographic plate, but the surrounding portion showed more or less radiating bands, which appear to coincide with depressions in the surface of the calculus.

No. IX. (*calculus* 121).—This consists of oxalate of lime followed by ammonium urate, and that again externally by mixed phosphates. In the specimen experimented with the central nucleus had fallen out and the space was bounded by a layer of ammonium urate. The only shadow obtained was that in the situation of the absent nucleus, and which corresponded to a layer of ammonium urate surrounding the vacant space.

No. X. (*predynastic Egyptian calculus*).—By the kindness of Mr. Shattock we were enabled to make an experiment with the predynastic Egyptian calculus preserved in the Royal College of Surgeons.[1] The calculus was found lying amongst the pelvic bones of a boy in a predynastic grave, computed to date from at least 5000 B.C. The external part consisted mainly of phosphates, with a certain amount of urate, and the nucleus of alkaline urate. The nucleus was detached and placed upon a plate in a similar manner to the calculi obtained from our own museum; it was found to produce no effect. The more external portion, owing to its extremely irregular shape, was only in contact with the plate at one or two points and over a small surface. The area of contact between the plate and the surface of the calculus shows a local deposit of silver.

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[1] A full account of this calculus (in which the presence of the chitinous capsules of bilharzia ova was looked for with a negative result) was published by Mr. Shattock in the *Transactions of the Pathological Society*, 1905, lvi., p. 275.

SUMMARY.

(1) The compact nucleus, which consisted of comparatively pure uric acid, produced no effect.

(2) The maximum effect was produced by the more porous parts, which consisted mainly of urates with an admixture of uric acid.

(3) The phosphatic portion gave little or no effect, except when mixed with urates. This fact is noteworthy in connection with the suggestion that the reduction of silver depends upon a slow oxidation of organic matter.

(4) A predynastic Egyptian calculus yielded a positive result with the external portions and none with the nucleus.

(5) The silver of the film is not reduced if the calculus is separated from it by a thin sheet of mica.

A detailed account of these and further experiments in the same direction will appear in the forthcoming report from the Cancer Research Laboratories of the Middlesex Hospital.

Pathological Section.

February 18, 1908.

Mr. S. G. Shattock, President of the Section, in the Chair.

Acidosis in Pregnancy.

By J. B. Leathes.

The subject which I have been asked to bring up for consideration is one that has as yet not been fully worked out, but the work that has been done has brought to light facts and raised questions that may be of the greatest importance in some of the graver disorders of pregnancy. There are three papers or groups of papers in which, in Germany and America, attention has been called to the significance of the high proportion of the excreted nitrogen that may take the form of ammonia salts in the urine during pregnancy. The first of these is the work of Zweifel [20]. He found in eighteen successive cases of eclampsia that the nitrogen excreted as urea formed a smaller proportion of the total nitrogen than is the case under normal conditions (from 27 to 70 per cent., instead of about 87 per cent.), while the ammonia nitrogen was high (in one case as high as 16·5 per cent., the normal quantity on a normal diet amounting to not more than 5 per cent. and commonly even less than 4 per cent.).

Zweifel has interpreted these figures in ways that are not altogether in harmony with current views, and more particularly ascribes the high percentage of ammonia to the excretion of lactic acid, which he regards as the cause of the eclampsia. He finds lactic acid commonly in eclampsia both in the urine and in the blood. In three cases he found more in the placental blood and in three cases more in the fœtal blood from the umbilical cord than in the maternal blood. On these grounds he thinks the acid is produced in the child and causes poisoning of the mother and is not produced in the mother as a result, for instance, of the fits. Again, in four cases the lactic acid was found in the urine before the fits, and similarly in two cases in the blood. The figures obtained in one of his cases are as follow:—

	Weighing	Lactic acid in milligrammes	Per mille
Placenta	386 ...	181 ...	0·7 of blood reckoned as two-thirds the weight of the placenta
Blood of umbilical cord	95 ...	24·5 ...	0·26
Maternal blood ...	390 ...	32·5 ...	0·08
Urine	820 ...	415·0 ...	0·5

Now lactic acid has been repeatedly found in the urine of epileptic patients. Inouye and Saiki [11] examined the urine passed one or two hours after epileptic fits on several occasions in each of seven epileptic subjects and found lactic acid every time, and in much higher concentrations than Zweifel, up to 1 per cent., or twenty times the amount in the case quoted above. In the intervals between the fits lactic acid could not be found, so that the conclusion drawn is not that the acid is the cause of epilepsy, but that it is produced in the muscles as a consequence of the impeded respiration. There is much evidence that this is the consequence of deficiency in the supply of oxygen to the muscles. After removal from the body the muscles, it is well known, produce lactic acid, but this is not the case if a free oxygen supply is maintained; and if lactic acid has been formed the excised muscles, when supplied with oxygen, are able to cause its disappearance [10]; or if in a living animal the circulation through the hind limbs is interfered with, the venous blood from those parts is found to be charged with lactic acid. It is true that normally the urine contains no detectable amount of lactic acid, and normal blood at most a trace; but 0·3 per cent. was found in a case of normal but protracted labour [3]. It is also necessary to remember that the estimation of lactic acid in urine and blood is very imperfect. If lactic acid in known amount be added to blood and the amount estimated the result is, it may be, 50 per cent. too low, and with urine the results are even less satisfactory still [3], so that it is not possible to attach very great significance to differences between the amounts found in blood from different sources when the amounts are so small. And even granted that the amount in the fœtal blood is sometimes larger than that in the maternal, this is no less than might be expected from the interference with the fœtal circulation liable to occur during delivery.

But in any case lactic acid is a substance that it is difficult to look on as likely to cause profound intoxication. The injection of sodium lactate into the circulation of dogs in amounts far greater than any ever found by Zweifel causes no signs of disturbance; his highest figure is 0·11 per cent., and in the experiments referred to the amount injected was sufficient to give a concentration of 1 per cent., or even 1·5 per cent. in the blood [4] [5].

But, whether the lactic acid is significant or not, the high percentage of ammonia in the urine observed by Zweifel is remarkable, and in some of the disorders of pregnancy considerably higher percentages still have been observed. Thus Williams [19] reports on three remarkable cases of severe vomiting in pregnancy in which the ammonia excreted in the urine accounted for 32, 35 and 45 per cent. respectively of the total nitrogen. Williams found on examining the liver of a case which was clinically precisely similar to these, central necrosis of the lobules resembling that found typically in acute yellow atrophy; and he refers to another case clinically and histologically identical with this one. He therefore regards these as cases of toxæmic vomiting allied to yellow atrophy, and this latter as a fulminating variety of the same disorder. Other cases of severe vomiting in pregnancy in which the ammonia coefficient is normal may be recognised by that sign as of a comparatively innocent type—cases of neurotic or, it may be, reflex vomiting. But when, he says, this coefficient is increased to 10 per cent. or 15 per cent. it would seem that the diagnosis of toxæmic vomiting is justified and an urgent indication given for the prompt termination of the pregnancy. The following is a brief abstract of his three cases of toxæmic vomiting, with the total nitrogen excretion of twenty-four hours and the percentage ratio of the ammonia nitrogen in the several specimens examined.

TABLE I.—WILLIAMS'S CASES.

Case 1. Vomiting began in second month; became incessant in third month; lost 20 lb. in weight. Pulse 132. Liver dulness 1½ in. above costal margin. No albumin nor casts.

	T. N. g.	N. as NH_3 per cent. of T. N.
Urine for twenty-four hours before abortion ...	4·5 ...	32·0 ×
,, one month after	12·7 ...	9·6

Table I.—Williams's Cases—*(continued).*

Case 2. Vomiting and indigestion at beginning of second month; incessant for three weeks in third month; lost 15 lb. to 20 lb. Liver dulness normal. No albumin nor casts.

	T. N. g.	N. as NH_3 per cent. of T. N.
In twenty-four hours urine	6·0	16·0
	7·5	20·7
	5·5	35·5 ×
	8·0	32·0
Abortion	6·4	24·0
	9·5	18·0
	8·7	19·0
Vomiting returned fifth day	7·0	25·5
	5·5	21·0
	4·7	12·0
Went out tenth day against advice	4·5	19·0
One month later	—	4·0

Case 3. Vomiting incessant through second month. Liver dulness normal. No albumin nor casts. No jaundice. Post-mortem not obtained; but a similar case showed central necrosis of liver lobules as in acute yellow atrophy.

	T. N. oz.	N. as NH_3 per cent. of T. N
In twenty-four hours urine	7·5	15·8
	9·7	15·3
Abortion	16·8	19·0
	9·0	31·0 45·5 × 24·0 36·0

Death forty-eight hours after operation.

Much the fullest and most valuable set of analyses of the urine in pregnancy is that given by Ewing and Wolf [6]. In addition to the determination of the total amount of nitrogen in the urine, and the amount of that which was present in the form of ammonia, they estimated also the urea, uric acid and creatinine, and consequently were able by subtraction to form an idea of the amount of nitrogen not accounted for by any of these substances. Exact data have been obtained by Folin in precisely this way for a number of normal individuals taking a diet of milk and eggs containing 19 g. of nitrogen. These average normal figures are given for comparison in the first line of the table that follows, which is an abbreviated version of those given in Ewing and Wolf's paper.

Their cases are divided by them into six groups. The first two groups exhibited no abnormal symptoms, but while the urine from Cases 1 and 2 gave normal or almost normal figures, the second group, Cases 3 to 6, showed signs of the abnormal distribution of the excreted nitrogen, which is, in varying degrees, characteristic of the remaining groups.

The next six cases, group C, were clinically alike in that they were all characterised by severe vomiting lasting for a considerable time in the early months of gestation. In the urine of all of these the urea nitrogen forms a low percentage of the total nitrogen. In two-thirds the ammonia is high, in one case very high; but in all six the undetermined nitrogen is uniformly high even in the two cases with normal ammonia coefficients.

The fourth group of cases were clinically different from these; although vomiting was commonly a pronounced feature in them, too, it came on for the most part later in pregnancy, and was accompanied by albuminuria and other signs of renal failure, high tension pulse, indigestion, temporary blindness, headache, or œdema. These cases of pre-eclamptic toxæmia gave somewhat different figures. The ammonia coefficient was comparatively low, if not normal, and only on one occasion in one of the cases reached double figures. Nevertheless the amount of urea was too small, and for the reason that the undetermined nitrogen was in excess. In two-thirds of the cases more than 20 per cent. of the nitrogen of the urine occurred in compounds of undetermined nature on some of the days for which the data are given.

The fifth group is composed of eight cases of eclampsia, of which five recovered and three were fatal. Here, too, the tables show that the output of ammonia was frequently not excessive—in half the cases it did not reach double figures. And yet though the undetermined nitrogen was higher than normal it formed more than 20 per cent of the total nitrogen in only one of the cases, which case recovered. In another case that died the figure would certainly have been high had the analyses been complete. The last group of four cases of acute yellow atrophy showed similarly irregular figures for the distribution of the excreted nitrogen. But the cases in both these two groups were not sufficiently long under observation for the analyses to have the value which attaches to those carried out on the cases in the third and fourth groups.

TABLE II.—EWING AND WOLF.

	Total Nitrogen g.	Urea Nitrogen percentage of T. N.	NH_3 Nitrogen percentage of T. N.	Undetermined Nitrogen percentage of T.N.	
Normal values (Folin)	19	85–88	3–5	4–6	
A.—Normal values in pregnancy (E. & W.):					
Case 1	4–9·7	74–84	3·5-5·8	8–14·8	
,, 2	—	71–84	2·8-5·8	7–14	
B.—Abnormal values with no symptoms:					
Case 3	6–7·4	63–67	6·2-8·6	15	
,, 4	8	54	11	24	Severe constipation.
,, 5	4–9	67–73	5·2-7·6	13-18·5	
,, 6	7·5–9	70–76	4–7	11–16	Fourth pregnancy; eclampsia with first pregnancy; casts at end of second.
C.—Toxæmic vomiting:					
Case 7	12·5	67–71	14·2-15	13	Persistent vomiting for two weeks; acetone.
,, 8	6·2	72	12	14	Persistent vomiting for four weeks, second and third month.
	—	80	5	16	Four days after abortion.
,, 9	6·5	60	10	18	Persistent vomiting for eight weeks.
,, 10	4·8	67	2·6	22	Persistent vomiting many weeks; jaundice; no acetone; constipation; "neurotic."
,, 11	3·3	47	*21*	24	Persistent vomiting; amaurosis.
Four weeks later	—	18	*43*	26	Acetone in breath; starvation.
	2·2	32	*38*	28	Labour induced (twins).
Two weeks later	—	77	5·5	13	Well; nursing.
Case 12					
Two days before	4·3	79	2·3	13	Vomiting from conception; abortion at three months; death twenty-five days later.
Seven days after abortion ...	2·8	70·5	7	18	
D.—Pre-eclamptic toxæmia:					
Case 13	—	65	6·7	28	Second pregnancy; high tension pulse; indigestion.
	—	56	8·2	16	Abortion, seventh month.
	5·5	74	7	10	Third pregnancy, sixth month.
	6·3	75	4	16	Neuralgia, high tension.
,, 14	9·6	60	10	27	Jaundice; vomiting; high tension pulse.
Five weeks later	—	74	6	14	Condition improved; labour normal four months later.
Case 15	8	62	9	19	Œdema; headache; indigestion; pulse > 100.
,, 16	9·6	65	4	25	Vomiting; high tension; jaundice.
,, 17	5	80	5	10	Acute nephritis.
,, 18	8	68	4	21	Vomiting; high tension; jaundice.
,, 19	16	73	4·4	17	Vomiting; high tension; jaundice; headache.
	2·7	62	5	28	
,, 20	—	73	6	16	Marked œdema; urticaria.
,, 21	—	58	5·8	33	Headache; amaurosis; œdema; vomiting.

TABLE II.—EWING AND WOLF—*(continued).*

	Total Nitrogen g.	Urea Nitrogen percentage of T.N.	NH_3 Nitrogen percentage of T.N.	Undetermined Nitrogen percentage of T.N.	
E.—Eclampsia :					
Case 22	8·7	70	5·3	19	Five convulsions ; coma ; no acetone.
,, 23	18	67	7	22	Six convulsions ; coma ; no acetone.
	11	78	8	11	Delivered.
,, 24	4·7	73	9	11	Five convulsions.
,, 25	—	42	18		Twenty-five convulsions ; coma.
	9·9	79	8	8·5	Delivered.
,, 26	3	68	8·7	15	Delirium ; jaundice ; very high tension pulse ; *no convulsions.*
	8·6	80	7·5	10	Day after delivery.
,, 27	—	36	6	—	Convulsions ; coma.
Next day ...	—	43	14	—	Died ; liver, early thrombosis and autolysis.
Case 28	1·7	62	10	14·5	No acetone ; one fit ; coma ; collapse ; death.
,, 29	4·5	65	14	13·6	Thirty convulsions ; liver, early thrombosis and autolysis.
F.—Acute yellow atrophy :					
Case 30	11	58	4·5	[19][1]	Vomiting to fifth month ; amaurosis ; œdema ; stupor ; convulsion ; coma till death on thirteenth day ; liver extensively necrosed.
Three days later	8	68	17	[13]	
Case 31	—	26	16	[22]	Coma and collapse in sixth month ; liver autolysis ; no necrosis.
,, 32	7·2	64	9·3	[12·5]	Nursing for fourteen months ; no liver dulness.
,, 33	7·2	79	10	[10]	Vomiting one week ; jaundice ; delivery, followed by coma and death in forty-eight hours ; liver necrosed.

[1] Two days after delivery.

It is in the very full and often extended observations of the urine in the third and fourth of the above groups of cases that the great interest of this work lies, for though the cases with one exception were not actually fatal they were obviously serious, and in some instances would no doubt have ended fatally if the pregnancy had not been terminated.

The two types of abnormality are fairly distinct. One is characterised by severe vomiting in the early months with high ammonia coefficients and generally large amounts of undetermined nitrogenous substances, but no renal affection. The cases described by Williams emphasise the gravity of this condition, described as toxæmic vomiting, and with their very high ammonia coefficients give further significance to this abnormality of the urine. The other is an affection of the later months, in which the vomiting is secondary to renal complications, a pre-eclamptic condition in which the urine does not commonly have a particularly high ammonia coefficient, though the undetermined nitrogen appears to be very abundant.

The question raised is: How far are these abnormalities in the urine diagnostic indications of the gravity of the disorder? Do they lie at the root of the malady, as early fundamental symptoms of a disturbance of metabolism, of which, it may be, yellow atrophy or conditions of that order of gravity on the one hand, and eclampsia on the other, may be merely the final climax? If that is so, of course the importance of exact analysis of the urine in pregnancy is obvious, and Williams may be right in suggesting that an ammonia coefficient of 10 per cent. or more should perhaps be taken as an indication for the termination of the pregnancy.

High ammonia coefficients are well known to occur in a number of conditions. In the following table (III.) are some figures obtained by Folin [8] from normal individuals, in the first line on a diet of eggs and milk, rich in nitrogen; in the next four lines on a diet of arrowroot and cream, poor in nitrogen but otherwise sufficient. In these latter the figures given are the lowest observed in each subject for the urea and the highest for ammonia and undetermined nitrogen, the diet having been taken for five days or more in each case. The ammonia is high and also the undetermined nitrogen. It is true the absolute amount of both these forms of excretory nitrogen is smaller than on the full nitrogen diet: 12 per cent. of 3·5 g. = 0·42, while 5 per cent. of 16 g. = 0·8. But then the total nitrogen excretion of most of the patients examined by Ewing

TABLE III.—FOLIN.

	Total Nitrogen g.	Urea Nitrogen percentage of T.N.	NH_3 Nitrogen percentage of T.N.	Undetermined Nitrogen percentage of T.N.
Diet I.—Eggs and Milk N. = 19 g. Average excretion of several subjects	16·0	85—88	3—5	3—6
Diet II.—Starch and cream N. = 1 g. Four subjects Maxima or minima	3·8	60	13·6	12·8
	3·6	62	11·3	10·9
	2·7	61	11·4	11·6
	3·5	59	9·4	15·4

and Wolf was also very low. In Case 11, when the ammonia coefficient was 21 per cent., the total nitrogen amounted to only 3·3 g., so that the total NH_3 was 0·69. And in these cases, besides a low intake of proteid, the supply of food in other forms was, owing to the vomiting, also low, which was not the case in Folin's subjects on the starch and cream diet. Now starvation is another of the conditions in which not only high coefficients but large absolute amounts of ammonia are observed. This is illustrated by the figures given in Table IV., taken from the results of the examination of a professional faster by Cathcart [1], and the figures given by von Noorden [17], from a patient treated for gastric ulcer by complete starvation, are similar (Table V.). In one case by Nebelthau of starvation in hysteria the ammonia coefficient reached the figure of 66 per cent. of the total nitrogen.

TABLE IV.—STARVATION (CATHCART).

	Total Nitrogen	Urea Nitrogen percentage of T. N.	NH_3 Nitrogen percentage of T. N.	Undetermined Nitrogen percentage of T. N.	Absolute Amount of Nitrogen as NH_3
Average of four days on Diet I. (Milk and eggs)	16·0	87	3·5	5·7	0·6
Fast—Fourth day	13·7	82	9·5	3·3	1·3
Sixth day	10·8	78	12·6	3·3	1·35
Eighth day	9·5	71	14·9	6·8	1·4
Fourteenth day	7·8	77	9·5	4·6	0·7

TABLE V.—STARVATION: VON NOORDEN'S PATIENT (GASTRIC ULCER).

Day	T. N.	NH_3	Percentage
1st	10·2	0·8	8
2nd	11·8	0·9	8
3rd	9·9	1·4	14
4th	8·6	1·5	18

That vomiting and diarrhœa in children cause high percentages of NH_3 has been pointed out especially by Czerny and Keller, who found commonly figures varying from 30 to 52 per cent.; and it is well known, too, that it is not necessary that starvation should be complete in order to bring on the condition in which a large proportion of the nitrogen is excreted as ammonia. The abstention from carbohydrate food has the same effect, and it is possible that diabetes, itself the most familiar of all the conditions in which high ammonia coefficients (up to 40 per cent. and more) are common, is in this respect merely a special case of this defect; what carbohydrate is admitted into the body may in diabetes be excreted without combustion, and the same result is attained as if none were taken at all.

In all these conditions the excess of ammonia appears in the urine because the acids produced in metabolism, failing to find fixed alkalies to neutralise them, are neutralised by ammonia that would otherwise have been converted into urea. The acids may either be derived from the oxidation of sulphur or phosphorus set free from broken-down proteid, or may be organic acids that in disordered metabolism—in diabetes, for instance—or in deprivation of carbohydrate food, with or without complete starvation, have to be excreted because they are not oxidised to carbonic acid. Carbonic acid can be removed without loss of bases, whereas the unburnt organic acids must be excreted as neutral salts, and so carry out with them fixed alkali; and it must be remembered that acidosis, if by that we mean merely the excretion of excessive amounts of ammonia that would otherwise be converted into urea, may be due to a shortage of fixed bases as much as to an excess of acids. The salts of organic acids in ordinary diets supply the fixed bases required for removal of sulphuric and phosphoric acids as well as any organic acids that may escape combustion, and a diet poor in such salts must lead to an output of ammonia that would otherwise have appeared as urea. This is probably the explanation, at least in part, of the high NH_3 coefficients in Folin's subjects on a diet which, though plentiful, consisted only of starch and cream (Table III. *supra*). Similarly, too, in an experiment of Landergren's [12] a diet was taken for five days consisting of butter, 250 g.; oil, 190 g.; wine, 170 g. Table VI. gives the figures obtained. The high ammonia coefficient on the fifth day is due not only to the excretion of β oxybutyric acid resulting from the deprivation of carbohydrate food; another contributing factor was the

TABLE VI.—LANDERGREN.

Diet consisting of butter, oil and wine only.

Day	Total nitrogen	Urea N. g.	NH_3 N. g.	= per-centage of T. N.
1st	7·1	5·5	0·4	6
2nd	8·5	6·9	0·8	10
3rd	8·8	6·4	1·4	16
4th	7·1	4·2	1·9	25
5th	5·7	2·7	2·1	37

very small intake of fixed alkali, which could have been present in this diet only as salts of organic acids in the wine.

This same factor, deficiency of alkali, comes into play in some degree in prolonged vomiting, severe diarrhœa (when the stools may be actually alkaline), and in diabetes even, in which the loss of magnesium and calcium due to the prolonged drainage of bases by the unburnt organic acids, especially β oxybutyric acid, has been frequently noted. It may even be the case that the pathology of acidosis turns upon this drainage of bases from proteids and substances of a similar complexity, the needs of which for fixed bases in the proper execution of their functions are not to be measured by the comparatively slight acid character stamped upon them. They are commonly also basic in character as well, and in the absence of fixed alkali may be hampered in their activities by having to play the base to each other's acid.

Another possible significance that may be attached to an increase of the ammonia in the urine is associated with the function of the liver in converting ammonia and carbonic acid into urea. The effect of removing the liver in birds is to cause two-thirds of the nitrogen to be excreted as ammonia, and in dogs Nencki and Pavloff found that after Eck's operation the ammonia in the urine rose till it formed 20 per cent. of the total nitrogen contained in it. The association of acute yellow atrophy with pregnancy, and of the lesions in the liver characteristic of that disease with the cases of toxæmic vomiting described by Williams, have been sometimes regarded as indications for attributing the abnormalities of the urine in pregnancy to disturbance of hepatic functions. But there is less justification for supposing that disease of the liver is likely to lead to a diminished formation of urea than is often admitted. In severe cirrhosis of the liver Weintraud found normal figures for the ammonia coefficient commonly in all but the final stage, and though cases are on record where higher values have been found (Hallervorden, 1·4 to 2·5 g. per diem; Mörner and Sjöqvist, 2·4 g. per diem 9·5 per cent.; Fawitzki, five cases, ranging from 7·6 per cent. to 17·5 per cent.; Schubert, one case, always more than 10 per cent. and once 17·5 per cent.), nevertheless Weintraud found that even when the coefficient was high the administration of ammonium salts led to an increased output of urea and not of ammonia, just as is normal. This, therefore, points to the excess of ammonia in the urine being due not to failure of the liver or other organs to convert it into urea, but to the presence of unburnt acids in the urine. So, too, in yellow atrophy itself, though, as in Ewing and Wolf's cases, there is generally an abnormal amount of ammonia, only one case is recorded, so far as I have discovered, where it amounted to more than 20 per cent. of the total nitrogen (Münzer 37 per cent). But Münzer, who studied the urine of a case of phosphorus poisoning in detail for several days, and found ammonia coefficients varying from 8 to 18 per cent., was able, by administering on two successive days 6 g. of sodium bicarbonate, to reduce the output on the first day from 16·6 per cent. to 11 per cent., and on the second to 6·2 per cent. This certainly seems to point to the high figures here, too, being due not to a defective formation of urea but to the excretion of acids that should have been converted into carbonic acid.

So far, therefore, as the ammonia figures in the disorders of pregnancy are concerned, before it can be safely maintained that these high figures are a sign in themselves of a toxæmia that is likely to prove fatal unless the most active measures be taken, it is necessary to prove that they are not sufficiently accounted for by some of the attendant circumstances of the patient's condition: the low nitrogen content of the absorbed food, the imperfect nutrition due to the incessant vomiting, the loss of alkali in the vomit, aggravated possibly by the requirements of the fœtus. The facts that the high figures may occur without any symptom of general disturbance (Ewing and Wolf's cases, 3 to 6), and that there may be profound disturbance with normal figures (*ibid.*, Cases 10 and 12, the latter fatal), are difficult to reconcile with such a view. In some of the recorded cases acetone is mentioned as having been detected, which is no more than could be expected, and, therefore, the acids associated with acetone were probably present in sufficient quantity to account for some of the excess of ammonia. The valuable data contained in Ewing and Wolf's paper, and the stray observations outside it, point to the necessity of decisive experimental work before their pathological significance can be positively defined.

The other abnormality of the urine, the large output of unknown substances containing nitrogen, is more striking and more suggestive, for this does not appear among the results of mere starvation, as the figures given by Cathcart (Table IV.) show. The only substances that have been detected giving rise to variations in the nitrogen in this category are amido acids, and though, of course, this does not warrant a prejudice as to the meaning of the figures in disorders of pregnancy, if the pressure of amido acids in abnormal amounts were proved, it is possible that some of the abnormal ammonia figures might be explained in that way, and that the tendency to associate the abnormality of the urine with disturbance of hepatic functions, and to regard it with suspicion as a sign of danger from that quarter, would be strengthened. It is to be remembered that Ewing and Wolf's first six cases, in which no symptoms were present, all showed high figures in this column, at any rate occasionally, so that it is not clear that great importance should be attached to these facts. On the other hand, it may be noticed that all the cases in which there is a note of jaundice occurring were cases in which particularly high figures under this heading were observed.

But it must be confessed that up to the present time such complete analyses as those given by Ewing and Wolf have been carried out for very few, if any other, pathological conditions, and till more is known of the kind of figures which other diseases present in this respect, and more than that, till the nature of the substances referred to as undetermined has been defined, it is hardly legitimate to speculate upon, much less to draw inferences from, variations in the amount of unknown substances, which may or may not be the same in any two cases. The only admissible justification for attempts to interpret this phenomenon will be definite work on the subject.

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DISCUSSION.

Dr. BEDDARD said that a distinction should be drawn between acidosis and acute acid intoxication. By acidosis was meant a condition in which an abnormal quantity of organic acids escaped oxidation and appeared in the urine. Such a condition might go on for months, or even for years, without producing marked symptoms or a fatal result, as in diabetes and acute starvation. But acute acid intoxication was a condition with which life was not compatible. Therefore, if serious symptoms were to be attributed to acidosis by lactic acid, a substance which is not toxic apart from its action as an acid, it was necessary to show that the acidosis led to acute acid intoxication of the body: with the possible exception of diabetic coma this has not been shown to be the case in any disease. With regard to the question of ammonia, this was present in the urine solely because it had to neutralise acids formed in the body. No matter how much ammonia a person had in his urine, if he were given a sufficient quantity of sodium bicarbonate by the mouth all of it, except a trace, would be got rid of. In many cases the acids were inorganic, and it was only when ammonia was oxidising organic acids that one should speak of acidosis. Folin's cream and starch diet, for instance, was one which, on oxidation, led to a considerable excess of inorganic acids over inorganic bases; but to call that acidosis was a misnomer, inasmuch as it was the neutralisation of inorganic, not organic acids. With regard to acidosis in liver diseases, Dr. Leathes had dealt with that very fully. Serious liver changes had often been described in association with eclampsia, and what was striking about the figures which the opener had exhibited was that, unless the liver disease was extremely severe and widespread, no acidosis, or very little, was present. And that, although in such a case as nearly complete removal of the liver or in acute yellow atrophy, there was considerable acidosis, and there might possibly even be acute acid intoxication of the body, though that was very doubtful, there was no reason to think that the acidosis caused the symptoms, because exactly similar symptoms were found in other liver diseases in which no acidosis was present. On the other hand, it was possible to have a very considerable change in the liver and yet no acidosis. For instance, in some recent experiments with poisoning by hydrazene there was no acidosis whatever, and yet that substance produced changes like phosphorus, with the exception that its action was specific upon the liver and did not involve the muscles as did phosporus. Again, by the injection of hæmolytic sera considerable areas of severe necrosis could be produced in the liver comparable to those in eclampsia, and again there was no acidosis. Therefore, it seemed to him, on a priori grounds, extremely unlikely that the acidosis which might exist in pregnancy could possibly be the cause of serious symptoms occurring, but was rather to be looked upon as the result of vomiting, starvation, &c.

Dr. LEATHES, in answer to Dr. Beddard, said he was glad to find that that speaker felt much as he did himself in the matter. It was impossible at this stage to say that the figures shown were fundamentally related to the causation of the disturbances ; they might be purely incidental and due to a variety of causes. He did not regard the high ammonia coefficients as a sign of true acid intoxication. It would be much more interesting if one knew the meaning of the high percentage of undetermined nitrogen which was present in the urine.

Dr. BAINBRIDGE said there was not much left for him to say on the subject, but one or two points in Dr. Leathes's remarks might be referred to. With regard to starvation accounting for the high coefficient of nitrogen, it was interesting to note that the ammonia coefficient could be considerably reduced by giving carbohydrate in pernicious vomiting, and that starvation did exist in pernicious vomiting could be shown by the very low nitrogen output and by the patient's wasting. But there were other factors which contributed to that high ammonia coefficient. Dr. Leathes referred to the retention of bases, and he (Dr. Bainbridge) thought that might possibly play a considerable part in the condition. It certainly influenced the ammonia coefficient in diabetes, because that coefficient in diabetes did not by any means correspond to the amount of acid which had been or was being excreted. And when the acid fell, as it might do under various conditions, the ammonia coefficient remained high for some time, and then fell much more slowly. That he believed to be due to the fact that the calcium and magnesium were now being retained, and the ammonia figure remained high, so as to diminish the loss of those bases : and he thought the same thing might happen in pregnancy, so that both those factors could influence the ammonia coefficient in pregnancy. If the subjects of pernicious vomiting were starving the probability was that their urine would contain acetone bodies, and such had been found, though they had not been very systematically looked for. They might to some extent influence the ammonia coefficient. Dr. Beddard had referred to the point he had intended to mention, namely, Jackson and Pierce's experiments with hæmolytic sera, in which serious lesions of the liver, closely simulating those seen in pernicious vomiting, produced no effect on the ammonia coefficient. But they found one fact which had a relation to the figures shown by Dr. Leathes, namely, that the undetermined or residual nitrogen was very high. That they attributed to autolysis, the products of the necrosis of the liver being excreted in some form other than either ammonia, uric acid or creatinine. And it seemed possible that some of that residual urinary nitrogen which was found in toxæmic vomiting might be coming from a necrosed liver, though he did not know that such was the case. With regard to the practical application of those figures, he could see no value in an isolated examination of the ammonia and total nitrogen in the urine in a patient suffering from pernicious vomiting ; and when one considered what a careful interpretation was required of a series of figures resulting from a complete analysis, it would be agreed that the facts were not yet ripe to justify the taking of any practical steps as a result of examination of the urine, and certainly not unless such examination were very complete.

Dr. LEATHES, answering Dr. Bainbridge, said that probably the high figures seen in the last columns of those tables did mean some proteid digestion products or autolytic products. The only things which were known to vary materially in that column of residual nitrogen were amido acids, such as alanine and glycocoll, which had been found in the urine in small quantities. And if it did mean an abnormally large amount of products of proteid autolysis, it was interesting in connection with what was found in the liver in some of those cases. He did not think anybody could yet say that that nitrogen was a product of proteid autolysis, nor yet what it was likely to be if it was not.

Pathological Section.

March 17, 1908.

Mr. S. G. SHATTOCK, President of the Section, in the Chair.

Three Specimens of the Larvæ of Eristalis tenax, passed by the Bowel.

By S. G. SHATTOCK.

Eristalis tenax: larvae per intestinum dejectae.

SUMMARIUM.

IN mulieris dejectis hae larvae in numero circa viginti ac viventes repertae sunt.

Herbae aquaticae *(Nasturtium officinale)* copiam aegra edere solebat. In hac forsitan tecta muscae ova ingesta erant.

Ad respirandum dubitari non potest quin aer in intestino inclusus, larvis sufficeret.

Casubus in haud paucis muscarum aliarum larvae *(Homalomyia* praecipue) in dejectis observatae sunt, sed larvarum Eristalis exempla vix ulla narrantur.[1]

Ad similitudinem apium maris musca ipsa *(Eristalis tenax)* prope accedit.

Haec similitudo ut ab auctoribus quibusdam indicatum est, modum explicat antiquum quo apes e bovis cadavere generari dicebantur. (P. Virgilii Maronis, Georgica, Liber iv.)

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[1] Wagner, B.: *Stettiner Entomolog. Zeit.*, 1870, xxxi., pp. 78-80; *Eristalis arbustorum.* Riley: *Insect Life*, 1888-1889, ii., p. 261, *Eristalis tenax*, *Eristalis dimidiatus.*

The specimens were sent to the College of Surgeons (in the Museum of which they have been placed) by Mr. H. McQuade, of Totton, Hants, with the statement that they had been passed by the intestine; two, which were in spirit, had been passed three weeks previously, the other on the morning on which they were despatched, this one being alive at the time. The donor remarks that their vitality was astonishing.

The patient suffered no inconvenience, although much horrified by the sight of the larvæ. She first passed about twenty or thirty at one time, without any "tail." After a few days interval the others came singly, and these were furnished with "tails." Full doses of santonin, and of mercury and other intestinal antiseptics were given. In answer to a letter of mine Mr. McQuade writes (January, 1908): "There is not the smallest doubt but the specimens were passed by the bowel; the whole length of time they were observed was about four weeks, and none have been seen since those which I sent were passed"; and in answer to a further inquiry as to the possible use of drinking water from other than usual sources (the larvæ being found in the muddy water of pools and ponds) the donor supplied the interesting statement that his patient had recently arrived from France, where she had eaten a good deal of watercress.

The larvæ, which were identified by Mr. E. E. Austen, of the British Museum (Natural History), are those of the dipterous insect *Eristalis tenax*.

Eristalis tenax; Linneus. "Habitat in aquis stagnantibus, cloacis bibliopagorum pappo,[1] vix preli pressione destruenda larva."

DIPTERA.

Family	...	...	*Syrphidæ.*
Subfamily	...	...	*Eristalinæ.*
Genus	...	...	*Eristalis.*
Species	...	...	*Tenax.*

The specimens comprise two larvæ and one pupa.

"The mouth of the larva is surrounded by a cartilaginous margin, enclosing a fleshy organ; the under side of the body is furnished with seven pairs of membranous feet, provided with small hooks. When full grown they quit the water and bury themselves in the earth; the body shortens and becomes a puparium, in which the organs of respiration consist of four small horns placed in the front part of the body."[2]

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[1] Bookbinder's paste, presumably.

[2] Westwood: "Classification of Insects."

The larvæ occur particularly in stagnant water and muddy places, and live at a considerable depth below the surface. Breathing takes place by means of a flexible tube at the posterior end of the body—the so-called tail of the "rat-tailed" larvæ. Oviposition occurs upon material at the surface or edge of water, the ova being about 1 mm. in length.

In the present case the ova were most probably taken by the patient concealed in watercress, and subsequently underwent development into larvæ in the intestinal canal. When it is remembered that sewers constitute one of the habitats of the larvæ there is nothing surprising in the presence of the latter in the contents of the intestine. The oxygen for respiration during their development would be furnished, one must believe, by the air naturally swallowed. Chemical analysis, in fact, of the intestinal gases reveals the presence of atmospheric oxygen as well as of nitrogen, the latter being mainly derived from the same source.

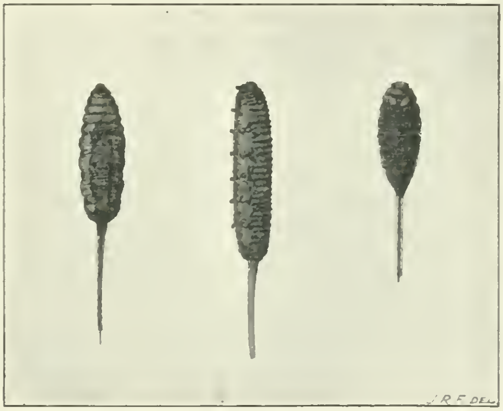

FIG. 1.

Three larvæ of *Eristalis tenax* passed by the intestine. The middle is viewed from the side and shows the seven pairs of short prolegs. That on the right has shortened, and is becoming transformed into a pupa. Each is furnished at the posterior end with a respiratory tube or "tail." From the end of two of the breathing tubes there projects a finer process which can be traced within the rest of the tube, and consists of an invaginated part of it, the tube, when fully extended, being considerably longer than in the condition shown. (Natural size.)

Eristalis tenax: larvae per intestinum dejectae. E corporis extremitate posteriori respirationis projicitur tuba, invaginata partim sive retracta. (Magnitudinis naturalis.)

The fly which is developed from the pupa has the general form and coloration of a drone, which it equals in size. It is, however, at once distinguishable from such by the fact that it has but two membranous wings, whilst the Hymenoptera have four, the posterior pair being smaller than the anterior.

A considerable number of cases of intestinal myiasis might be collected. The larvæ passed by the bowel, however, have been, in nearly all, those of different species of the dipterous *Homalomyia* (Syn., Anthomyia), a fly which in general character resembles the house fly.

As this particular subject does not strictly concern that under notice, it may be enough for me to refer to one of the earliest and one of the latest observations in our own literature. So long ago as 1789 a case of this kind was recorded in the second volume of the *Memoirs of the Medical Society of London* by Dr. W. White. The patient, a man, aged 30, passed a large number of maggots after taking a purgative for constipation. Some of these were kept in a box and developed into flies. A figure accompanies the article. The fly was probably *Homalomyia canicularis.* The most recent record of the same class is that by Baker[1] of the passage by the intestine of larvæ, which were diagnosed by Austen

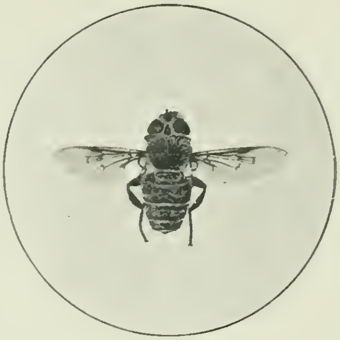

FIG. 2.

The fully developed dipterous fly of *Eristalis tenax*, showing its close external resemblance to a drone. It will be noted, however, that it is furnished with two wings only, the Hymenoptera being furnished with four. (Natural size: from a photograph.)

Eristalis tenax: Musca perfecte evoluta. Ad similitudinem apium maris accedit; alis autem duabus solum praedita est; quattuor praedita apis. (Magnitudinis naturalis.)

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[1] *Trans. Burma Branch Brit. Med. Assoc.*, January, 1892, i.

as those of *Homalomyia scalaris* and *Homalomyia canicularis*. As the species of the genus Homalomyia undergo their transformation in decaying vegetable matter, manure, and excrement, the development of ingested ova in the intestinal contents offers no difficulty. But no instance of the passage of Eristalis has yet been recorded in our own literature. The only previous example is one referred to in the work of J. C. Huber, "Bibliographie der klinischen Entomologie," iii., p. 12, Jena, 1899. This reference is to a very brief statement by Riley,[1] who mentions the fact that some larvæ of *Eristalis tenax* were received by him from Dr. J. A. Linter with the note that they had been passed by the intestine. Riley records, also, an observation of the passage of larvæ of *Eristalis dimidiatus* from the intestine of a young woman. Five specimens of a third species of Eristalis (*Eristalis arbustorum*) are reported by Wagner[2] as having been voided *per rectum* in the case of a female.

Of the family Syrphidæ, to which Eristalis belongs, Westwood (loc. cit.) remarks that many species so closely resemble humble-bees and wasps that they are constantly mistaken for them by the inexperienced. This general resemblance explains, as modern entomological criticism has pointed out, one ancient belief concerning the origin of bees. The method of raising "bees" from the putrefying carcass of an ox described by Virgil is aptly explained on the assumption that flies of the Eristalis species bred from ova deposited in the putrefying fluids of the carcass, or from ova in the intestines ingested by the animal before death, were mistaken for Hymenoptera. After discussing the loss of bees from disease, the Roman poet cites, as a method practised in Egypt, that of raising a new stock from the carcass of a steer which was allowed to putrefy at spring time, after the viscera and limbs had been well bruised, the skin being left intact.

> Interea teneris tepefactus in ossibus umor
> Aestuat, et visenda modis animalia miris,
> Trunca pedum primo, mox et stridentia pinnis,
> Miscentur, tenuemque magis magis aera carpunt.
> Donec, ut aestivis effusus nubibus imber,
> Erupere, aut ut nervo pulsante, sagittae,
> Prima leves ineunt si quando proelia Parthi.
>
> —*Georgicon*, Liber iv.

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[1] *Insect Life*, ii., p. 261. United States Department of Agriculture; Division of Entomology. Washington, 1888–1889.

[2] Wagner, B. *Stettiner Entomol. Zeit.*, 1870, xxxi., pp. 78–80

> The tainted blood in this close prison pent
> Begins to boil, and through the bones ferment.
> Then, wondrous to behold, new creatures rise,
> A moving mass at first, and short of thighs ;
> Till shooting out with legs, and imp'd with wings,
> The grubs proceed to bees with pointed stings :
> And more and more affecting air, they try
> Their tender pinions, and begin to fly,
> At length, like summer storms from spreading clouds, &c.
>
> — VIRGIL. *Georgics*, iv. (Dryden's translation, line 435).

Dryden does not profess to give a close translation, and it may be pointed out that he has gratuitously added stings to the flies, of which there is no mention whatever in the original.

The Influence of the Testis upon the Secondary Sexual Characters of Fowls.

By C. E. Walker.

(From the Cancer Research Laboratories, University of Liverpool.)

Several experiments upon the effect of castration (partial and complete) and of vasectomy in fowls have been recorded.[1] The results of these have shown that even when only a small portion of the testis is left after incomplete castration the secondary male sexual characters appear, and that the secondary sexual characters become fully developed in young birds or sheep after ligature and division of both vasa deferentia, the testicles being left in situ. In two cases the authors cited observed shrinkage of the comb after castration of the adult cock. They conclude from their experiments that the appearance of the secondary sexual characters is not due to metabolism set up by nervous reflex arising out of the mere physical function of the sexual mechanism, but is due to an internal secretion of some of the cells contained in the testis. In conducting a research not directly connected with sex, it became desirable to obtain some further information, if possible, as to whether or not the testis possesses any function which has an effect upon the organism as a whole apart from the production of spermatozoa. The evidence bearing upon this point referred to above and that of the well-known effects of castration upon young male animals seems to indicate that some functions which influence various parts of the organism before the time of puberty and at about that time are probably possessed by the testis as it is becoming mature.

The main points which it was wished to ascertain by the experiments here described were, whether any function or functions influencing the production of the secondary sexual characters are possessed by the testis throughout the period of sexual activity, and also whether the cells of the female organism possess the potentiality of differentiation usually found only in the male.

A fresh preparation of cock's testes was made as follows: The testes were removed and cut into small pieces. They were then pounded in a mortar, and the contents of the mortar strained through fine muslin with a small quantity of normal saline solution. As the pounded testes lying upon muslin stretched over a glass funnel were pressed and rubbed with a large pestle, practically everything passed through excepting a small amount of membrane. The amount of salt solution used was roughly about double the bulk of the testis. The strictest aseptic precautions were used, and the test tubes containing the mixture were kept in an ice chest. In spite of the fact that no preservatives or antiseptics of any kind were used at any stage in the proceedings, this preparation of testis was kept in one instance at room temperature for nine weeks without the slightest sign of decomposition occurring. Half a cubic centimetre of this preparation was injected subcutaneously into certain hens once a day. The results obtained with two adult hens were as follows:—

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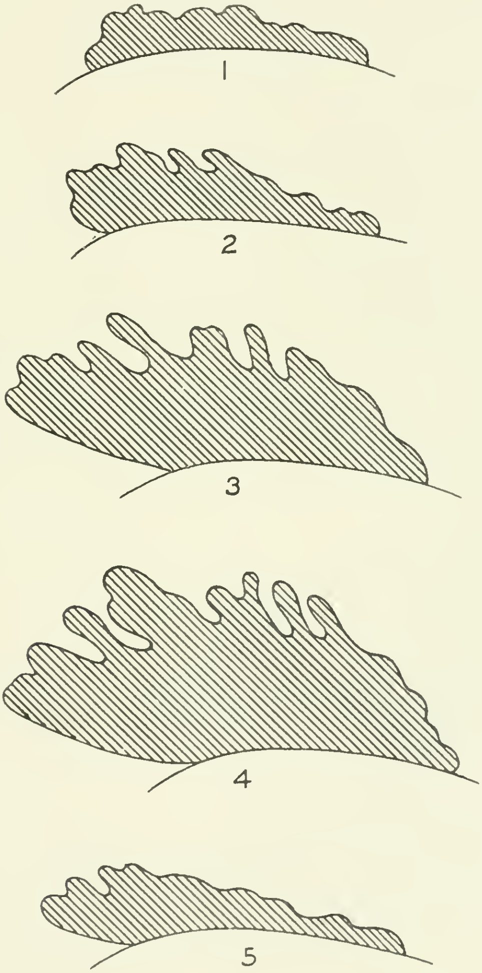
[1] S. G. Shattock and C. G. Seligmann: *Trans. Path. Soc. Lond.*, 1905, lvi., p. 57, and 1906, lvii., p. 69.

The accompanying diagrams refer to the outline of the comb of one of these hens only. As the same series of changes occurred in the other hen it has not been thought necessary to give the outline of its comb at the different periods. The outlines were made by cutting a pattern out of stiff cardboard so that it fitted round the comb as accurately as possible. The injections were commenced upon February 1, 1907, and fig. 1 represents the outline of the comb at that date. In a few days the combs and wattles of the two hens became much brighter in colour, and it soon became evident that they were growing rapidly. Fig. 2 shows the comb of the same fowl as that represented in fig. 1 as it was upon February 22. Figs. 3 and 4 represent the comb of the same bird as it was upon March 16 and June 30 respectively. The combs of both birds were of the most brilliant scarlet colour and were firm and apparently highly vascular. The wattles also grew considerably. From about the end of June the combs and wattles appeared to remain stationary. From August 30 until October 4 the hens were injected with only about half the quantity of the preparation of testis. On October 4 the combs and wattles were not nearly so brilliantly coloured. They looked slightly bluish, were slightly wrinkled, and appeared flabby. Their size was not, however, appreciably diminished. Since October 4 no injections have been given. Fig. 5 shows the outline of the comb of the same hen as is illustrated in the other figures as it was upon November 9. In both hens the combs and wattles had decreased to about the same extent, had a shrivelled appearance, and had become of a distinctive bluish colour. A careful watch was kept upon the spurs, but nothing sufficiently marked to be worthy of record was observed. It must be remembered, however, that the spurs of cocks normally take a considerable time to develop, and that the full injections were only continued for six months.

Some young hens have been treated in the same manner since they were eight weeks old. This second experiment, however, was only commenced on April 6, and though the results are entirely satisfactory they differ somewhat from those detailed above, and are not yet sufficiently complete for publication.



Outlines showing the increase in size of the comb of a hen treated with injections of a salt solution extract of cock's testicle. No. 1 represents the comb before the administration of the injections. No. 4 shows the maximum development of comb and No. 5 the retrogression after their discontinuance.

The two hens referred to in the detailed experiment were fully adult, and though their exact age is uncertain, both must have been aged over 2, and one of them probably aged about 4. They both laid eggs at intervals up to the middle of March, 1907, when they ceased, and laid none until January, 1908, when they began again, four months after the cessation of the injections. For the first three weeks after the injections were commenced they appeared quite friendly towards the cocks that were from time to time put into the greenhouse where they lived. After this, however, they began to attack the cocks when they were put in the house with them, and continued to do this in a more and more marked manner as time went on.

The results may be summarised as follows: While the preparation of fresh testis was injected into the hens their combs and wattles grew in size and became more brightly coloured, reaching a maximum in five months. The size and coloration of these appendages remained the same while the injections were continued after this maximum had been reached. When the injections were discontinued the combs and wattles shrank and lost their bright colour, and in the course of little more than a month were very little larger than they had been before the injections were commenced.

Although the experiment was made with only two hens the results would hardly appear explicable as a coincidence. Had growth in the comb and wattles only occurred in connection with the injections, such an interpretation might have been probable, but the shrinking of these appendages in both cases when the injections were discontinued seems to exclude the probability of a coincidence.

It would seem, then, that (1) the testis possesses some function which influences or controls the appearance of the secondary sexual characters, and that (2) the potentiality of differentiation necessary to the production of some at any rate of these characters is present in the cells of the female.

Photographs of the hens at different periods were taken, but they do not illustrate the growth of the comb so well as the outline drawings. This is owing to the fact that when the comb reaches a certain size it droops over to one side, and consequently does not show up satisfactorily in a photograph.

A Case of Congenital Defect of the Muscular System (Dystrophia muscularis congenita) and its Association with Congenital Talipes equino-varus.

By RUSSELL HOWARD.

THE case to be described was one of twins born at full time. The mother and father were healthy people and had eight children, all of whom are alive and healthy. The mother was aged 42, and there was nothing unusual about the pregnancy. The other twin was born dead, and is reported to have had some slight deformity of the feet, but this was so slight that it was not considered worth while to have it investigated, and the child was buried. The child under notice lived seven days.

External Appearances.—The length from the buttock to the crown of the head was 11 in. The arms were 6 in. and the legs 7 in. long. The head was well formed, and showed the moulding of a first vertex presentation. The circumference through the external occipital protuberance was 11½ in. The hair was well developed. The anterior fontanelle was widely open, but the posterior fontanelle was as closed as in a normal full-term fœtus. The ears were normal in appearance. The face was plump, and had the usual appearance of a newly born healthy child at full term, the nose, eyes, eyelids and mouth being normal. The body was well covered with fat and was plump, but a little shapeless and flattened from side to side, but the alterations were not sufficient of themselves to call for comment. The buttocks were flattened, and the usual anal cleft almost absent, the anus being very conspicuous and the perineum more prominent than usual. The scrotum and penis were somewhat flattened, and projected down below the flexed thighs. Both testes were in the scrotum, and the penis was well developed for a fœtus. The umbilicus was normal and the cord had separated.

Upper Extremity.—The arms were plump but not well formed, the roundness of the shoulder being absent and the whole arm being too cylindrical. The arm was internally rotated, so that the olecranon looked almost directly forwards, and the back of the wrist was to the front, the thumb being internal. The forearm was, however, in a position of semi-supination, so that the effect of pronation was produced by internal rotation of the shoulder-joint. The elbow was flexed and the wrist was flexed and adducted to the ulnar side. The thumb was slightly abducted. It was not possible to put the limb into the normal position without severe wrenching, and after moving it forcibly it always sprang back into the original position.

Lower Extremity.—The lower extremity was not well developed, although the amount of subcutaneous tissue seemed normal. The thigh was flexed at a little more than a right angle to the body, the leg was flexed at a right angle to the thigh, and the foot was in an aggravated position of talipes equino-varus. The internal malleolus was prominent, and on the outer surfaces of the knees were small depressed areas of skin looking as if pressure had been applied. It was not possible to straighten the feet without applying undue force, and they sprang back into the original position as soon as the force was removed. The two feet could be readily interlocked and fitted into one another, and it appeared as if they were so placed *in utero.*

Abdomen.—As regards the abdominal cavity there is little to be said; all the organs appeared to be perfectly normal, and presented the usual features seen in a newly born infant. The diaphragm macroscopically appeared normal. The crura were fleshy, and the muscular tissue was well marked. The central tendon appeared normal and the openings were in their usual position and of usual size.

Thorax.—The lungs were normal in appearance, but in a condition of atelectasis. The thymus was normal in size and position. All the chambers of the heart contained blood-clot and their appearances were normal. The foramen ovale was not completely closed, but there were no pathological changes in the various openings. The great vessels were normal. The sympathetic nervous system was normal in appearance to the naked eye, and the spinal nerves were as usual. No gross changes appeared in the veins, arteries or lymphatics. The œsophagus and trachea were normal.

Brain and Skull.—The cerebrum was somewhat soft and too decomposed to be available for section cutting, but on macroscopical examination it appeared to be normal. The convolutions were well marked. The ventricles were normal in appearance and contained only a small amount of cerebrospinal fluid, there being no suggestion of hydrocephalus. The cerebellum was normal in size and appearance. The mesencephalon and the medulla showed nothing abnormal to the naked eye. The membranes of the brain were normal and not adherent to the cortex. The various sinuses were normal, and the appearance of the cranial nerves and their mode of exit from the skull were as usual.

There was nothing in the brain or skull to suggest any abnormality. The spinal cord appeared perfectly normal to the naked eye. The membranes were normal, and the amount of cerebrospinal fluid was as usual. The spinal nerves were normal in appearance. No hæmorrhage had taken place into the cord or spinal canal, and cross sections at every level appeared normal. The chorda equina was normal and there was no spina bifida, the spinal canal being perfectly closed. It may be said that on gross examination of the central nervous system nothing abnormal was to be noted. It is to be regretted that microscopic examination was a failure.

Dissection of the Face.—The subcutaneous tissue of the face was normal, fat being present in the usual amount. The superficial muscles of expression, especially the orbicularis palpebrarum, the orbicularis oris, the zygomatic muscles and the platysma were all well marked and easily dissected. Both bellies of the occipito-frontalis were also as well developed as usual. The external appearances of the eye and the eyelids were normal, and a well-marked sucking pad was present.

Neck.—The sterno-mastoid was well marked, and had its usual origin and insertion. The upper fibres of the trapezius were normal, and the great muscles of the back of the neck, the complexus, semi-spinalis colli, splenius, capitis, &c., were as usual. The scalenes were normal in appearance, origin, insertion and relationship. The infra-hyoid group of muscles, including both bellies of the omo-hyoid, were normal in appearance and relationships. The intrinsic and extrinsic muscles of the tongue and pharynx were as usual. The cranial nerves and the nerves of the cervical plexus had their usual relationships and appeared normal in size. The muscles of mastication were normal and had their usual nerve supply. The arteries of the head and neck were normal.

Upper Extremity on Dissection.—The subcutaneous tissue appeared to be normal and there was a fair amount of fat. The subcutaneous vessels and nerves had their usual positions. The deep fascia was normal, not thickened, and readily dissected off the muscles. Lines of fat were found running between the muscles and also into them, so that in many cases accurate dissection of the muscles was not possible. The muscles appeared to be in various stages of fatty degeneration, and all were very small. Some, such as the biceps, were almost absent, and this was verified by the microscopic examination; whilst others, for example the triceps, the extensors carpi longior radialis and brevior, although small, showed less signs of fatty degeneration. It was especially difficult to follow the muscles to their origins and insertions as the tendons were markedly atrophied, but as far as they could be dissected all were normal in situation and relationship. The extensor communis digitorum tendon in particular was very small and atrophic, and could not be traced along the backs of the fingers. The clavicular portion of the pectoralis major was almost normal in external appearance, although smaller than usual. The amount of atrophy and fatty degeneration did not follow any particular nerve distribution, but appeared to be quite capricious, but no muscle could be considered quite normal in size or colour. The vessels were small, but had their usual branches and relationships. The brachial plexus appeared to be normal as regards the arrangement and distribution of its nerves, and the nerve relationships in the arm and forearm were as usual and the only abnormality was that the nerves were a little smaller than usual. The fingers presented nothing abnormal except the smallness of the tendons. The nails had reached the ends of the fingers.

Abdominal Muscles.—The rectus abdominis was pale and atrophied, and separated by about $\frac{3}{4}$ in. from its fellow of the opposite side, the gap being filled by a thin fibrous membrane. The external oblique, the internal oblique and the transversalis were all thin and badly developed, but to the naked eye were not infiltrated with fat. It was possible to separate the muscular portion of all three muscles, but their aponeuroses were blended into one. The quadratus lumborum was thin and small, but all the layers of the fascia lumborum were distinct and had their usual attachments. The internal and external abdominal rings were normal. All the abdominal nerves occupied their usual positions.

Muscles of the Back.—The latissimus dorsi had practically disappeared and could not be differentiated from the fat, but the erector and multifidus spinæ were easily demonstrated, and although small and pale showed no marked naked-eye fatty degeneration. Their attachments were normal.

Muscles of the Chest.—Both sets of intercostal muscles could be demonstrated. The serratus magnus was atrophied and infiltrated with fat, and the costal portion of the pectoralis major was in a similar condition. The pectoralis minor was small and pale but not infiltrated. The attachments of the abdominal muscles to the ribs were indefinite and difficult to demonstrate, the aponeuroses being very thin.

Muscles of the Lower Extremity.—The muscles of the buttock had almost disappeared, only appearing here and there as brown streaks amongst a mass of fat. Dissection of them was quite impossible, but from the situation of some of the tendons there was reason to believe that their attachments were normal. The hamstrings had their usual attachments, but were thin and fatty. The great sciatic nerve appeared normal. The anterior thigh muscles were atrophied, but their attachments to the sides and top of the patella were normal. The adductor muscles were atrophied but otherwise normal. The leg muscles were all small, but the calf muscles were less easily identified than the extensor group. The tendons were very thin, both on the dorsum and plantar aspects of the foot, but could be traced to their usual insertions. The small muscles of the foot did not appear to have suffered as much as the larger muscles, and the interosseous muscles were well marked. All the blood-vessels of the lower extremity were small, but had their usual relationships and branches. The sacral and lumbar plexuses, with their branches, were arranged in the usual way.

Joints: Upper Extremity.—The humerus was inwardly rotated as regards the scapula, and the articular surface was smaller than usual. The humerus itself was twisted in such a way that the lower articular surface, instead of looking antero-posteriorly, looked laterally, and the olecranon process of the ulna articulated on the internal aspect instead of the posterior. The radius articulated with the ulna in the usual way. The forearm was in the semi-prone position, and the superior and inferior radio-ulnar articulations were normal. The wrist was slightly flexed and in slight ulnar adduction, but otherwise the joints of the carpus and metacarpus were normal. The articulations of the phalanges were normal.

Lower Extremity.—The hip-joint could not be extended after all the muscles had been removed. On examination, the head of the femur was somewhat flattened, and at the upper part of the neck was an articular facet which corresponded with a facet in the upper part of the acetabulum and showed the place where the two bones had laid in contact owing to the flexion and rotation inwards of the femur. There was a marked degree of coxa vara, the great trochanter being on a higher level than the head of the bone; the neck was shortened. The great and small trochanters were not prominent, but had the usual relations and positions. After all the muscles had been dissected away round the knee-joint and only the ligaments left it was still impossible to straighten the joint. This was found on dissection to be due to two causes: (1) the shortening of the ligaments on the flexor surface of the joint; (2) the shape and position of the articular surfaces. The bones were only covered with smooth articular cartilage at the place where they were in actual contact, and the condyles of the femur articulated by two small oval facets, with similar facets on the tuberosities of the tibia, and the patella articulated with the femur by an entirely separate facet. The articular facets of the tibia were more widely separated than usual, and were so arranged that the tibia was internally rotated and sloped inwards at the knee-joint.

The foot was in a marked condition of equino-varus. The os calcis was so drawn upwards that it almost touched the posterior surface of the tibia, and the foot was turned inwards at right angles to the anterior aspect of the tibia and fibula. This inward twist was seen on dissection to be mainly, but not entirely, due to changes in the astragalus and the astragalo-scaphoid joint. The articulation of the astragalus with the tibia and fibula was almost normal. The neck was bent inwards so that the head looked inwards and the articular facet for the scaphoid was on the inner aspect of the neck instead of being on its anterior surface, so that the foot was abruptly bent inwards at the astragalo-scaphoid joint. The articular surfaces for the astragalus on the os calcis were also on the inner side instead of on the dorsum of the bone. The foot in front of the astragalo-scaphoid joint was nearly normal, but all the joints had a slight inward tendency and all the ligaments on the inner side of the foot were slightly contracted.

Sections for microscopic examination were made from the following muscles and groups of muscles: Gluteal muscles, hamstrings, anterior thigh muscles, lateral abdominal muscles, psoas, triceps, anterior muscles of the fore-arm, biceps brachialis, pectoralis major, scalenus anticus, and the tongue muscles, and control sections were made from the muscles of a normal fœtus.

Sections through the gluteal muscles showed a large amount of areolar tissue from which fat had been dissolved in the course of the preparation. The sections of the nerves, vessels and lymphatics of the part were apparently normal, and there was no perivascular or perilymphatic infiltration.

Here and there through the sections were bundles of muscular fibres which showed the following conditions: In some cases nothing but the muscle sheath remained, the whole of the contents having disappeared. Some fibres showed the sarcolemna, and inside this a little brown pigment and here and there a muscle fibre. In others the muscular structure was more definite, but the sarcous elements were broken up and the nuclei were abundant.

In places fibres showing a definite cross striation were seen and only to be distinguished from the normal muscle by their pale colour and slight want of clearness of outline.

Scattered amongst the degenerating muscular fibres were found some fibres of greater calibre than normal muscle fibres, resembling those seen on sections of muscles the seat of pseudo-hypertrophic muscular paralysis. The nerve endings in the muscles appeared normal.

There was no increase of the fibrous tissue of the part and nothing to suggest a chronic inflammation or a past acute inflammation.

Descriptions of the other sections would only be a repetition of the above. Some muscles, such as the biceps brachialis, the anterior thigh muscles and the calf muscles, were almost completely absent; whilst in others, such as the clavicular head of the pectoralis major, the changes were not so advanced, but in all degenerative conditions were present and no perfectly normal muscles were seen.

It is, however, to be noted that in every section fibres were to be seen that had definite transverse striation and which only showed slight signs of degeneration.

The muscles which showed the least degeneration were those of the head and neck; in fact, the naked-eye appearances were exactly counterparted by the microscopic sections.

Portions of all the important nerves, both spinal and cranial, were cut into microscopic sections and stained by Busch's method, hæmatoxylin and Van Gieson's stain, and hæmatoxylin and eosin; sections were also examined unstained and control sections were cut from a normal fœtus and stained in a similar manner. No differences were to be made out between the nerves from the case under discussion and the nerves from the normal fœtus, and one could certainly exclude any gross peripheral lesion.

It is to be regretted that it was not possible to get sections of the central nervous system, but the naked-eye appearance revealed nothing abnormal.

A consideration of this case shows that the only abnormality discovered in the specimen was a defect in the voluntary neuromuscular system, the remainder of the fœtus being apparently normal; and the point first to be discussed is whether the lesion is in the muscles or primarily in the nervous system. Unfortunately there is no microscopic report of the central nervous system, but the naked-eye appearances are normal. The peripheral nerves showed no lesion and the nerve endings in the muscles were normal.

It is difficult to imagine a lesion of the motor cells in the cord with such unchanged peripheral nerves, and it is unlikely that a cerebral degeneration would have neither naked-eye changes nor degenerative changes in the peripheral nerves. It is specially to be noted that the muscles supplied by the cranial nerves had almost entirely escaped.

It may fairly be considered that the primary lesion lies in the muscular system, and it may be one of the following three conditions:—

(1) A degeneration of the muscular system following acute inflammation or a chronic myositis.

(2) A non-development of the muscular system.

(3) A degeneration of the muscular system.

As regards the first of these conditions the microscopic appearances are antagonistic to the theory that the change is due to inflammation. There is no fibrosis, but merely a fatty degeneration, and there is no perivascular infiltration with small round-cells. The tendons are small but not adherent to their sheaths, and, in fact, in neither macroscopic nor microscopic appearance is there the slightest suggestion of inflammatory change. There is also much against the second theory of a non-development of the muscles. As far as can be ascertained all the muscles are present and have their usual origins and insertions, and some are well developed and almost normal. Where the muscular tissue is absent in many places fat replaces it and preserves somewhat the outline of the muscle, whilst in other places the muscle, although small and thin on macroscopic examination, looks almost normal on microscopic examination. The changes observed in the muscles also exactly follow the changes seen in the muscular dystrophies of post-natal life; in fact, it would be impossible to distinguish sections of these muscles from some sections of a case of pseudo-hypertrophic muscular paralysis.

This case may therefore be considered as a case of primary degeneration of the muscles occurring before birth. This condition has been described under the name of dystrophia muscularis congenita, and is recognised clinically.

The object of this preliminary communication is to draw attention to the condition of the joints, and more particularly of the joints of the foot. The surfaces of the bones on which articular cartilage is found corresponds exactly with the places where the bones are in contact, and it is obvious that the places of contact determine the condition and shape of the joint surfaces. In a normal fœtus which moves its limbs freely *in utero* the joint surfaces are extensive and adapted to the various movements, but in the fœtus under consideration movements must have been absent or very limited, and as a consequence the joint surfaces are limited to the places of contact, and, as has been shown, these surfaces are situated in abnormal positions.

In cases of congenital talipes equino-varus not due to obvious nervous lesions, such as spina bifida, the articular covered surfaces are also abnormal in position and shape, particularly that on the head of the astragalus, which looks inwards and closely resembles the facet on the astragalus in this case. The muscles and ligaments also are contracted in a similar manner to that described above. If in the case described the muscular lesions had been limited to the legs it would have been labelled clinically as a case of congenital talipes equino-varus.

Dr. Hutchison has allowed me to examine and report on a case of a deformity occurring in the upper extremities only, in which the upper limbs were in a similar position to that of Erb's paralysis, that is, in a precisely similar position to the upper extremities of the case under discussion. The joints were fixed in a similar manner as that described, and there was the same want of muscular development and action. The lesion has remained limited to the upper extremity and shows no sign of progression.

I have also seen a child, aged 9, who had both lower extremities in the position of talipes equino-varus and both upper extremities in the position of Erb's paralysis, and whose only active muscles were those of the trunk, head and neck. The disease was not progressive. The specimen described is obviously only an advanced degree of the condition present in those cases, and the child probably only died because the abdominal and thoracic muscles were involved, rendering respiration difficult, though not impossible, as the child lived seven days. It is interesting also that the twin child was reported to have its feet in a position of equino-varus, but otherwise appeared normal. It is also well known clinically that in cases of congenital talipes equino-varus the muscles generally do not develop, although the position of the foot is corrected and the patient is able to walk well. These cases are mentioned and figured in every orthopædic text-book, and every surgeon is familiar with the wasted spindle-shaped legs of these patients, although the patient stands and walks flat on the soles of the feet. This non-developed condition of the muscles is always apparent as soon as the excessive subcutaneous fat of infants disappears.

An examination of the muscles in a case of congenital talipes equino-varus shows them to be small with small tendons, but otherwise normal, in exactly the same way as some of the muscles of the specimen were; in fact, the condition seen in this fœtus is merely an advanced stage of the condition found in congenital talipes equino-varus.

If it be conceded that congenital talipes is due to a limited dystrophia muscularis congenita, the non-development of the muscles after removal of the deformity is explained, and also the marked differences met with in response to treatment in these cases, some readily responding to treatment and reduction of the deformity being easy, others requiring very prolonged treatment and the ultimate result being very unsatisfactory.

It is also desired to call attention to another deformity of the foot, the explanation of which is unsatisfactory, which passes under the names of pes arcuatus, pes plantaris, pes cavus, and others. It has been ascribed to anterior polio-myelitis of the short muscles of the foot and to contraction of the tendo Achillis, to the wearing of high-heeled shoes and to various other causes. The deformity usually starts in early life (5 to 8) without any discoverable cause, affects one or both feet and is associated with wasting of the muscles of the legs. The deformity is progressive, the foot assuming the position of talipes equino-varus, but it may be spontaneously arrested with any degree of deformity.

There is no reaction of degeneration in the muscles and no sensory changes, and the disease is not a family one.

I have elsewhere given reasons for believing that the disease is a primary muscular dystrophy, and I would suggest that it is allied to dystrophia muscularis congenita, and I hope at a further date to bring other evidence to support this view.

The Relations of Endothelioma to other Forms of New Growth.

By W. S. Lazarus-Barlow.

The form in which the present communication is brought forward depends upon the fact that the investigations upon which, largely, it is based have been carried out at my instigation at the cancer research laboratories of the Middlesex Hospital. The papers that will be read by Messrs. Courtauld and Leitch and Mr. Rowntree immediately after these opening remarks consequently form integral parts of one whole, and for each subject with which he will deal each gentleman is individually responsible. It has been thought well, however, that a short general survey of the scope of their researches should precede their remarks.

When examining a large number of microscopical sections of new growth from all parts of the body, and when conferring with other pathologists upon some of the more difficult types of growth, two facts become clear. The first is that in most situations which are pre-eminently the seats of special varieties of growth (*e.g.*, cervix, breast, tongue), certain specimens depart so widely from the common type for the part that they can only be forced into a common class with violence. The second point is that there is the greatest uncertainty as to the type of growth which should be called an endothelioma, some persons even going so far as to say that they have only seen one or two during an examination of tumours extending over a considerable number of years.

If we start from first principles it is clear, assuming that endothelial cells on occasion proliferate to the formation of a new growth, that an endothelioma can arise from the endothelium lining a lymphatic or a blood-vessel or one of the large lymphatic spaces, such as the pleural cavity. It is further clear that if such abnormal proliferation of endothelial cells take place it can either be directed towards the lumen of the tube from which it originates or away from that lumen, or in both of these directions. If proliferation take place inwards the accumulated cells will ultimately fill the lumen of the tube; if outwards, the lumen of the tube will persist, but the wall of the tube will be composed of a number of layers of endothelial cells instead of possessing merely a single layer. If proliferation of the endothelial cells take place both inwards and outwards a picture composed of the two mentioned will be produced. On this assumption one ought to meet with growths that may fairly be termed "entheliomata," "peritheliomata" and "perientheliomata."

In a paper read to the Glasgow Medico-Chirurgical Society in February, 1907, upon endothelioma, and published in the *Glasgow Medical Journal*, I suggested that certain varieties of new growth, met with fair frequency in the cervix and breast, and usually termed respectively squamous- and spheroidal-cell carcinomata, are identical in appearance, and are explicable on the assumption that they are examples of perientheliomata. One important common feature of these growths is the presence of a number of larger or smaller round or oval lumina, which may be empty or contain degenerated material. Drs. Courtauld and Leitch have made these lumina the subject of special investigation with particular reference to the method of their formation. One of the points which I would wish to submit to your consideration is the significance of these appearances in forming a diagnosis. Since it is seen that they are formed after one or other of the only two methods by which it is known that normal channels are formed in the embryo, they appear to me to be fairly comparable with ordinary blood or lymph channels.

The second point of difficulty concerns certain varieties of growth which I have ventured to call "Malpighian or basal squamous-cell carcinomata." I believe that a class of squamous-cell carcinomata exists which consists of pure basal cells, both in the primary growth and in metastases, without any admixture of the ordinary prickle or keratinising layers of the skin. It follows from the last that "cell-nests" are characteristically absent, and that a diagnosis from spheroidal-cell carcinoma on the one hand and a growth of the entheliomatous type on the other will be difficult. I therefore suggested to Mr. Rowntree that he should investigate the question of prickles in this connection. Clearly, if prickles are to be found between the cells of the basal layer in ordinary skin and between the cells of the so-called pure Malpighian carcinomata, a means of identification of the latter would be obtained and the possibility of their confusion with entheliomata would be avoided. Mr. Rowntree's investigations show that such a distinction can be made.

Considering the new growths of the cervix and breast on these lines, I believe that about 10 per cent. of all growths in the two situations which are at present diagnosed as carcinomata—whether squamous or spheroidal—belong to a single class practically identical in appearance in the two situations, and having the following characteristics: They show larger or smaller alveoli, containing cells which are provided with a relatively large amount of protoplasm, a relatively small nucleus which contains little chromatin heaped in the centre and connected by a few fine strands with a thin layer of chromatin that lies at the circumference of the nucleus. They show the presence, with greater or less regularity, in the alveolar masses of cells of a number of lumina, sometimes only the large variety, sometimes only the small or "secondary" variety, while sometimes both varieties are present. And, further, the characteristics above mentioned are produced with greater or less fidelity in any metastases that may be formed.

Even if it be granted that the criteria that have been given are sufficient to differentiate the growths in question from the ordinary carcinomata of cervix and breast, it is necessary to determine whether lumen formation occurs in the case of undoubted spheroidal- and squamous-cell carcinomata, since it is conceivable that it might only be a change comparable to the colloid change or the keratinising change, for example, and is not an indication that the growth originates from a fundamentally different variety of cell. Dr. Courtauld has therefore investigated a number of undoubted spheroidal- and squamous-cell carcinomata to determine whether lumen formation occurs in them. Since we know that a growth originating in cells which normally line a tube (*e.g.*, columnar-cell carcinoma of the rectum) preserves that tubular arrangement, whereas a carcinoma arising from cells which normally do not line tubes (*e.g.*, squamous-cell carcinoma of skin) does not form tubes but solid alveoli of cells, it seemed reasonable to assume that new growths in which lumen formation is present originated from cells which normally line tubes or cavities, and hence that the lumina are indications of a function of the cell which, though deficient, is not entirely lost. Dr. Courtauld's work shows that the formation of lumina in the types of growth mentioned, though met with, is uncommon and, in the case of squamous-cell carcinoma, is excessively rare.

Turning now to the question from the point of view of endothelium, the first consideration is the phylogenetic origin of that variety of cells. In the ninth volume of the *Archives of the Middlesex Hospital* Dr. Courtauld investigated this subject, using very early placental tissue for the purpose and considering the mode of formation of the endothelium lining the newly forming blood capillaries. He proved, as I think conclusively, that the endothelial cells originate from the loose connective tissue of the chorionic villus and that epiblastic layers take no share whatever in the process. It follows from this that an endothelioma must be classed along with the mesoblastic growths and side by side with the sarcomata, whatever its histological appearances may be. Further, if a type of growth originate from cells which are essentially mesoblastic, and (from the accidental circumstance that they normally line tubes and spaces) resemble a surface or a glandular epithelium, it is not unlikely that that growth will possess more or less unstable characters, in one case veering more towards the mesoblastic or sarcomatous type, in another to the epiblastic or carcinomatous type. It is in this way that I would explain those tumours which are said to consist of both sarcoma and carcinoma, such as the tumours of the uterus shown at the meetings of the Pathological Society of Great Britain and Ireland by McWeeney and by Teacher, and the case of mouse tumour believed by Ehrlich and Apolant to have become altered into or to have caused the origination in the tissues of the host of a spindle-cell sarcoma. Unless we are to discard entirely our present views of the differentiation of the blastoderm into epiblast, hypoblast and mesoblast, with the entire pathological superstructure that has been built thereon, it is inconceivable that a carcinoma can become converted into a sarcoma, and, in our present state of knowledge at least, it is as difficult to imagine a carcinoma causing a sarcoma to originate in the tissues of the host. On the other hand, if we consider growths arising from the inner surface of the dura mater, the pia-arachnoid, the pleura and the peritoneum, we find a diversity so great as to range from an intensely cellular growth that might be mistaken for a spheroidal-cell carcinoma to a dense fibrous-looking growth that might be mistaken for a fibro-sarcoma or even for a non-malignant fibroma.

In this connection it is necessary to draw attention to the enormous degree to which purely physical conditions can alter the appearance of cells. In the section of an ordinary polypus which is placed under the microscope the epithelial layers of the skin close to the base of the polypus have undergone a change which can be followed with the greatest ease, and which has resulted in the formation of a layer of spindle-cells as typical in appearance as any found in a spindle-cell sarcoma. In this specimen it has unfortunately been impossible to determine whether prickles persist between the fusiform cells. If, then, it is possible for cells of so great a relative degree of stability as those of squamous epithelium to undergo an alteration in appearance so striking, it is clear that it is in the highest degree unsafe to assume in the case of a new growth that alteration in appearance of cells, however profound, is indicative of a fundamental change of their nature. Indeed all who are as well acquainted with new growths in their metastatic appearances as they are in their primary appearances are aware that a certain amount of latitude must be allowed to the rule that new growths "breed true." Nevertheless, there is a considerable difference among growths with reference to the degree to which metastatic nodules differ in appearance from the primary growth. Thus the typical squamous-cell carcinoma, columnar-cell carcinoma and all the varieties of sarcoma show a relative amount of constancy, while the spheroidal-cell carcinoma, if we except from that category the forms of growth which I am bringing before your notice and am suggesting should be regarded as endotheliomata, also shows a great similarity of the metastatic to the primary growths. In the special class under consideration, however, there is not as great a degree of breeding true. Although it is common for lumina to be found in the metastases, they are not generally so large or well formed; the amount of protoplasm surrounding the nucleus is smaller than in the primary growth, the nucleus itself often shows a more diffuse staining of its chromatin, while an arrangement of the cell masses very similar to, but as indefinable as, that which obtains in the case of rodent cancer is often met with. In view of the great variability which obtains amongst growths originating in connection with endothelium in various parts of the body, to which reference has already been made, this variability of appearance in the metastases of the growths under consideration is intelligible upon the view that they are endotheliomata.

An important side issue of the question is that of the position in histological nomenclature which we should assign to the new growths met with in mice, with the associated question as to whether these growths are rightly to be regarded as carcinomatous. It may be conceded at once that in spite of certain differences that mark them off sharply from malignant new growths in man, they are true neoplasms. It appears to me, however, that the great majority conform more closely to the type of growth which I am bringing before your notice than to growths which can without doubt be classed as spheroidal-cell carcinomata. Indeed, Jensen's tumour might be taken as an example of the kind of growth the existence of which in man I am trying to demonstrate. The presence of lumina, sometimes very well marked, sometimes ill-defined, the character of the cells and the nuclei, the rodent cancer-like arrangement of the cell masses, the wide range of variability itself of these different features, are all met with in the human class of case, and are distinguishable from the opposite conditions which obtain in the groups of definite human carcinoma and definite human sarcoma. Just as typhoid was at one time confused with typhus fever, so I submit that the growths in question, whether in man or in mouse, are being confused with the carcinomata. One important consideration bound up with the question is the great difference in malignancy that will characterise the members of the group. It is only in the rarest instances that an endothelioma of the dura mater forms metastases, whereas the type of mammary and cervical growth which I am bringing forward is characterised by a great tendency to form metastases; endotheliomata of the pleura and peritoneum occupy an intermediate position, forming few metastases, as a rule, in distant parts, but enormously widespread growth locally. And since the liability to the formation of metastases, whatever that may mean in essence, is closely bound up with the danger of the particular type of growth to life, it becomes necessary to determine, if possible, whether the tumours in mice are to be approximated to that variety of endothelioma in man which kills essentially or to that which, so to speak, kills accidentally. Until this point is settled we cannot be certain whether investigations on mouse tumours have a direct bearing upon the entire range of human carcinoma or only upon a subsection which, even if the view I am advocating be conceded, amounts to about 10 per cent. of carcinomatous cases.

To sum up, the propositions which I submit to the meeting are as follow:—

(1) That from their phylogenetic origin, combined with the method of arrangement of normal endothelium, endotheliomata must be expected to show a great variability of appearance, ranging between that presented by a typical spheroidal-cell carcinoma on the one hand and a typical sarcoma on the other.

(2) That the group of endotheliomata in man must be enlarged to include certain growths of the breast, cervix, &c., which have hitherto been considered as carcinomata.

(3) That the variable appearance of certain growths of the uterus in man, and of certain mouse tumours on transplantation, is best explicable on the view that such growths are examples of endothelioma.

Finally, though I am convinced that the mammary and cervical cases to which I direct attention constitute a well-marked group, I recognise that my suggestion that they originate from endothelium only amounts to a probability. In view, however, of their resemblances to growths which there is reason to believe have definitely sprung from endothelium, I hold that that probability is a considerable one.

The Occurrence of Lumina in Malignant Tumours.

By L. Courtauld and A. Leitch.

This communication deals solely with the formation of spaces or "lumina," as they have been called, in the cell masses of malignant new growths, a feature regarded by some as constituting a criterion for the recognition of a particular type of tumour. In the course of this investigation several varieties of lumina were found. Of these the largest and most conspicuous occurs not infrequently in cancers of the breast and cervix uteri, and occasionally in cancers of other tissues. A lumen of this type appears as a round or oval space of considerable size—up to $\frac{1}{4}$ mm. in diameter—lying in the centre of the larger cell masses. This space may be empty or may contain a certain amount of granular material, in which are embedded a number of small darkly staining nuclei. But perhaps the most conspicuous and characteristic feature of this variety of lumen is that it is lined by a well-defined single layer of flattened cells, which are surrounded by and continuous with the polygonal cancer cells composing the mass. To study the development of these lumina serial sections were cut of tumours showing this formation, and the course of events could thus be followed. It was then found that these lumina are formed in the malignant cell masses by a necrosis of the central cells. The first sign of their appearance is a loosening and dissociation of these cells, which lose their regularity of outline and their brilliancy of staining. Passing on through the series of sections this change becomes more pronounced and affects more and more cells until an irregular space, partially filled by granular debris, is formed in the centre of the mass. Eventually we arrive at a stage where a large lumen lined by flattened cells is seen; and still further on this gradually diminishes in size and finally ends bluntly in a condition exactly similar to that by which it commenced. A lumen of this nature can be followed through a great number of sections, forty or more, and, if reconstructed in plan, would appear as a fusiform channel lying in a column of malignant cells. It seems, therefore, that the condition is produced by a fluid necrosis, that the flattening of the cells lining the lumen is due to the pressure of the contained fluid, and that the granular debris consists of degenerated cancer cells, and does not contain leucocytes as has been suggested.

Lumina of another variety are frequently seen in malignant growths of such glandular organs as the breast, and they are particularly common towards the growing edge of the tumour. These lumina are quite small, with a diameter equal to that of two or three cells only, and they cannot be followed through more than a very few sections. The cells surrounding them have a radial arrangement with the nuclei at the attached margin. In some cases it is possible to trace their formation by the union of the tips of proliferating processes of malignant cells springing from a glandular epithelium. It may therefore be assumed that this arrangement is an indication of an attempt on the part of the cancer cells to imitate the parent structure.

Finally there remains an entirely different type of lumination occurring in a very rare and peculiar variety of breast tumour. These tumours are highly cellular, but the great majority of the cells are completely degenerated, their nuclei have disappeared, and they stain with eosin alone. The only cells which have escaped degeneration are confined to a narrow zone surrounding some well-marked channels, with the walls of which they are in very close connection. Some of these channels are vascular, inasmuch as they contain blood. Others are empty, and may be either vascular or lymphatic; they do not appear to be glandular. Tumours of this nature are extremely uncommon; only three have been found among several hundred examples of cancer of the breast.

The Significance of Prickle-Cells in the Identification of New Growths.

By Cecil Rowntree.

The present investigation was carried out with a view to determining how far the presence of prickle-cells may be regarded as a characteristic feature of tumours originating in connection with squamous epithelium, and what bearing, if any, this may have upon the question of the identification of new growths generally.

Prickle-cells occur not only in the skin but in the mucous membrane of the mouth, tongue, pharynx and œsophagus, the vestibule of the nose, the anus, in the male the prepuce, glans penis and part of urethra, and in the female the vulva and vagina. Another situation in which they may be demonstrated is the skin lining dermoid cysts of the ovary.

As to the distribution of the prickle-cells in the epithelium of these regions, they are, of course, best developed and most obvious in the intermediate layer of cells, the so-called prickle-cell layer. But examination of thin and well-fixed sections—preferably stained with orange G—shows that prickles are present between all the cells, except the degenerated cells of the surface layer. The basal cells are closely packed, and the prickles in this situation are short and ill-developed, but they may usually be seen except on the lower surface of the cell, when they are replaced by minute dentate processes which serve to fix the epithelial cell to the underlying connective tissue.

The fact that prickle-cells are of such constant occurrence in squamous epithelium indicates that they constitute an integral part, and are characteristic of epithelium of this type; and, further, that an epithelium which in general appearance approaches the squamous type, yet contains no prickle-cells, can hardly be regarded as a true squamous epithelium. Such an epithelium, for instance, as that covering the prominent and exposed part of a nasal polypus—although composed of several layers of more or less flattened cells—cannot be regarded as an example of metamorphosis of columnar to squamous epithelium unless the presence of prickle-cells be demonstrated.

This constancy in the presence of prickles would lead one to expect that a malignant growth starting from squamous epithelium would also possess them, and this is the case, for it is found that the distribution of prickles in a typical squamous-cell carcinoma is very much the same as it is in normal skin. And this is true not only for the primary growth but for metastatic deposits in lymphatic glands or viscera.

In the case of a new growth showing keratinization there can be no doubt as to its origin from squamous epithelium, but there are certain forms of new growth in which there is no horny change, and it is in these that the presence or absence of prickles should be of importance.

Theoretically it is possible for a neoplasm to originate in the basal layer of cells and to retain throughout its growth and in its metastases the characteristics of these cells, and as the basal cells possess prickles we might expect this variety of growth to possess them also. We occasionally find malignant growths which conform to this type. The constituent cells are of somewhat columnar shape, and possess oval nuclei staining somewhat deeply; there is no keratinizing or other degenerative change, and prickles are present throughout the tumour.

When the basal-cell carcinomata arise in a skin surface it undoubtedly happens that they are sometimes regarded as rodent cancers, which differ from them by possessing no prickles and by the possession of smaller cells with rounded nuclei, arranged in small masses and columns.

There is another class of tumour which differs markedly in appearance from typical squamous-cell carcinoma, although it arises in situations such as the tongue, upper jaw, and cervix uteri, in which the latter is the common type, the first and most striking point of difference being the absence of keratinization. They are not basal-cell carcinomata, for prickles are characteristically absent. They possess moderately large cells arranged in large cell masses, many of which show a large central lumen, which Drs. Leitch and Courtauld have shown to be due to degeneration of the central cells. The nuclei are pale and differ from the nuclei of squamous cells by the arrangement of the chromatin. These characteristics are so constant and so striking that it is reasonable to suppose that this particular type of new growth may be of a nature entirely different from squamous-cell carcinoma and have a different origin.

Against this view it may be advanced that they are simply examples of squamous carcinoma in which growth has taken place in a manner so different from what is usual that the ordinary characters of squamous cells have not had the opportunity of developing, and that the presence of prickles is therefore not an absolute criterion of a squamous cell; in other words, that we have to deal with growths in which a certain amount of metaplasia to a less highly differentiated type of cell has occurred. But if this be so there remain to be accounted for certain tumours arising in the mammary gland which are histologically identical with the tumours of the tongue and cervix already described, and differing so considerably from the ordinary spheroidal-cell carcinoma usually occurring in the breast that it appears reasonable to place them in the same class.

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CONTENTS.

October 23, 1907.

PAGE

By Professor E. Goldmann :—

(1) The Growth of Malignant Disease in Man and the Lower Animals, with special reference to the Vascular System 1

(2) The Value of X-rays in the Diagnosis of Obscure Abdominal Cases ... 14

November 12, 1907.

An Analysis of 274 additional cases of Removal of Goitre by Operation. By James Berry, F.R.C.S. 21

December 10, 1907.

A Case of Complete Gastrectomy. By B. G. A. Moynihan, M.S. (Pathological Report by Dr. Craven Moore) 81

Cases of Cholecystectomy. By John D. Malcolm, F.R.C.S Ed. 93

February 11, 1908.

Coxa valga (Collum valgum). By A. H. Tubby, M.S. 107

March 10, 1908.

PAGE

On the Possible Uses of Lumbar Puncture in the Treatment of Otitic Meningitis. By ARTHUR E. BARKER, F.R.C.S. 143

May 12, 1908.

Sciatica and its Surgical Treatment. By J. CRAWFORD RENTON, M.D. 167

The Treatment of Ulcerative Proctitis by Zinc Kataphoresis. By F. C. WALLIS, F.R.C.S., and W. IRONSIDE BRUCE, M.D. 176

June 16, 1908.

Reduction of an old Subcoracoid Dislocation of the Humerus by Excavating the Glenoid Cavity through a Posterior Intermuscular Incision. By W. G. SPENCER, M.S. 187

Cystic Tumour of the Suprarenal Body successfully removed by Operation, with Notes on Cases previously published. By ALBAN H. G. DORAN, F.R.C.S. 201

By RUSHTON PARKER, F.R.C.S. :—

(1) Case of Severe Compound Fracture of the Arm 221

(2) Two Cases of Ununited Fracture treated by planting Small Fragments of Bone between the Ends 232

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Surgical Section.

October 23, 1907.

Mr. GODLEE in the Chair.

The Growth of Malignant Disease in Man and the Lower Animals, with Special Reference to the Vascular System.

By Professor E. GOLDMANN,

OF FREIBURG.

ON two previous occasions I have had the honour of addressing meetings in London in connection with work referring to the relation of cancer to the vascular system. In coming before you to-day, I do not wish to travel over ground already known to you. It is my main intention to give you a short *résumé* of the whole extent of my work, and at the same time to draw your attention to methods of investigation which I am sure will prove beneficial to all willing to co-operate with me in the solution of a problem so intimately associated with the biology of malignant growths. My work has been conducted on human and animal material. I owe a debt of gratitude to Professor Ehrlich, of Frankfort, and to Dr. E. F. Bashford, of London, for valuable material derived from the mouse, such as carcinoma in its various forms, sarcoma, mixed tumours and chondroma. In the short time at my disposal I can only mention my general results. A detailed account of, and an exhaustive reference to, the literature of the subject will be found in a forthcoming treatise. The points of view from which I have approached my subject are the following: (1) How far is the vascular system responsible for the dissemination of malignant growths? (2) What are the general conditions of circulation in these growths? and (3) What purpose does the multiplication of blood-vessels in malignant growths serve—merely that of nutrition or also that of defence?

As to the dissemination of tumours along the vascular system, I may refer to a treatise of mine published several years ago in which I proved, by means of an elective stain for elastic tissue, that both in the early stages of sarcoma, as well as carcinoma, an extensive invasion of tumour cells into the coats of blood-vessels occurs. It is a striking fact that this happens far more frequently in veins than in arteries, and again that the results of this invasion can only be traced locally, rarely exceeding the area of round-cell infiltration. Exactly similar results have been obtained in the case of carcinoma and sarcoma of the mouse. In examining mouse tumours, we must always bear in mind that we are dealing with vessels of comparatively small dimensions, whose elastic coat is hardly comparable with that of man. And yet careful investigation reveals in tumours of mice all the various stages of vascular degeneration common to man. I place such stress on these conditions as my extensive study of benignant growths, even of those verging upon the boundary of malignancy—for instance, goitre—has proved to me that tumour-cell infiltration into the walls of channels bounded by elastic tissue is a feature characteristic of malignant tumours only. I may add that, in those cases in which carcinoma of the mouse invades the lymphatic glands, the pathological condition of these glands is a counterpart to that found in man. The cells enter the marginal sinus and penetrate thence along the lymphatics into the medullary substance of the gland. It seemed to me of great interest to enquire into the conditions under which the tumour cells enter into the vascular coats. At a first glance it appeared most probable that they travel along the lymphatics. Yet all anatomists concur in declaring that the vascular coats contain no lymphatics, a statement confirmed by evidence gained from pathological experience, especially in cases of backward transportation of tumour cells along the lymphatic channels. In these cases the cells are almost exclusively confined to the perivascular spaces, and hardly ever penetrate the vascular walls. Hence we are bound to assume that the dissemination of tumour cells into the vascular coats is effected by blood-vessels. This seems all the more likely when we consider how different are the appearances of arterial and venous cancer, and how the distribution of tumour cells in arteries and veins coincides with that of the vasa vasorum. In arteries the tumour cells rarely proceed further than the outer coat, whereas in veins they are generally found beneath the intima. Thus arterial cancer appears as a form of periarteritis, venous cancer as one of endophlebitis carcinomatosa. Now it is accepted on all sides that the vasa vasorum in arteries remain within the limit of the outer coat, rarely branching into the superficial layer of the middle one, whereas in veins they extend beyond the middle coat into the region of the intima. It is a remarkable fact that this question, which is of vast importance in the pathology of the vascular system, has met with such scanty attention in recent years.

In order to gain experience of my own, I made use of a series of injections performed on fœtuses, bodies of newly-born children, and on amputated limbs removed from individuals of various ages and for various reasons, such as injuries, tuberculous joint or bone disease, and senile gangrene. I applied a method which furnishes us with ideal preparations, and found that in fœtuses and newly-born children there is no material difference between arteries and veins as regards their nutrient vessels. The distinction previously mentioned is found in the first year after birth and prevails as long as the vessels remain intact. Hence arteries perform their important functions with a minimum supply of nutrient vessels. This explains the fact that we may dissect out an artery completely from its surroundings without affecting its nutrition, and again, that the artery retains its marvellous power of healing although cut off from its original base. The blood supply of the arterial wall explains another and almost more important fact, viz., that, as Virchow puts it, the arteries act as isolators of pathological processes. All this changes as soon as pathological conditions arise in the artery, from within or without. Then the connective tissue, and, above all, the vasa vasorum, begin to proliferate, and finally we find vein and artery alike permeated throughout their whole breadth by numerous vascular channels. Such conditions are most common in vessels within the area of malignant growth, and it stands to reason that such vessels are more likely to harbour the tumour cells than healthy ones.

We must next consider whether these degenerative conditions in the vascular system are the causes of secondary growth. I believe that our views as to metastasis have been much modified by recent experience, which goes to prove that, both in man and animals, tumour cells pass into the circulation even at an early stage of the malignant growth. Lubarsch, Borst and others are therefore right in declaring that we must clearly distinguish between embolism and metastasis in malignant growths, as so many of the tumour cells are destroyed within the circulation before they establish secondary tumours. The conditions under which these arise are most complicated, and probably depend upon a number of factors, partly of a chemical nature, hitherto quite unknown. The question which has given rise to much discussion is how these cells get into the vascular system. There can be no doubt that vascular degeneration, such as I have described, is a predisposing factor, and yet Martin B. Schmidt has proved almost to a certainty that in his remarkable cases of abdominal cancer, in which diffuse embolism of the pulmonary arteries took place without the appearance of secondary lung tumours, the cells entered the blood-vessel through the thoracic duct. I am inclined to believe that we gain a more definite conception of the whole question of metastasis by paying due consideration to recent research work bearing on acute wound infection. Besides others I have, especially, Nötzel's work in mind; he has proved, in a series of experiments conducted on rabbits, that, after injection of bacteria into the knee-joints, these bacteria appear within a few minutes in the regional lymphatic glands and in the general circulation as well. The fact that these experiments were performed on healthy joints without any damage to them, and also without undue pressure of the injected material, allows of one conclusion only—that the absorption of the injected bacteria was effected by the lymphatic system. The rapid dissemination of the germs in these experiments appears to clash with the current theory of lymphatic circulation, inasmuch as the interposition of lymphatic glands has always been regarded as a retarding factor. Nötzel justly reminds us that, even in the lymphatic glands, anastomoses exist between afferent and efferent vessels, and that, according to the extensive investigations of Druner, such anastomoses are a most common occurrence among the larger lymphatic ducts. This again concurs with our surgical experience, which has shown that transverse ligature even of the thoracic duct has no ill effect upon the lymphatic circulation. On this basis we fully understand how germs travelling along the lymphatics enter the vascular system rapidly and without necessarily passing the lymphatic glands. We must, therefore, cease to regard the lymphatic glands as local centres of defence or as filters. All these facts have, in my belief, a most important bearing upon the problem of tumour metastasis.

As regards the relations between the lymphatic and vascular systems, I am inclined to assume that they are far more intimate than we have hitherto believed. It has struck me when injecting veins, such as the jugular, which are encompassed by lymphatic glands, that the injected fluid passes with the greatest ease into the glands, from which I infer that the vascular systems of gland and vein are closely associated. There must be relations of an intimate nature between the two systems.

I will not dwell upon the subject of hæmolymph glands, which are said to occur in man, but I wish to draw your attention to recent embryological research. Serbin, of the Johns Hopkins University, has shown that in man and mammalia of a high order the whole of the lymphatic system is a derivative of the veins, not only of the jugular but also of the iliac veins. Numerous instances have been recorded by anatomists of the older school in which great lymphatic trunks have been found to enter into other than the jugular veins, such as the iliac, azygos and others. Unfortunately, Henle's refusal to accept these observations has debarred others from following up more closely the question whether such connections actually exist, or whether they are to be regarded as exceptional. Not until all these dubious points are perfectly cleared up can we hope to understand the question of metastasis in general, and of tumour metastasis in particular.

I now arrive at my second point: What are the general conditions of circulation in malignant growths? For information on this score we are obliged to go far back into the history of medical science, back to the writings of John Hunter, Schroeder van der Kolk, Broca and others. In recent years Ribbert has been the only one to deal with this question, which he does in a short paper in which he endeavours to prove that the deficiency of blood-vessels in carcinoma is the cause of cell necrosis. In order to elucidate this point more precisely, I have applied different methods for the human individual and the mouse. In cases of human cancer, I exposed the femoral artery and injected an emulsion of bismuth and oil into the blood-vessels. After tying the afferent and efferent vessels of the cancerous organ, I dissected them most carefully from their surroundings and exposed them to the X-rays. These specimens are incomplete from an anatomical point of view, yet they give us a general idea of the state of vascularisation in cancer. This is well demonstrated in my plates taken from cases of carcinoma of stomach, liver, and other organs (figs. 1 and 2).

On examining these plates, the first point that strikes us is that the regular distribution of blood-vessels is disturbed by the invading growth. We know from the splendid work which Mall and his pupils have done that the distribution of blood-vessels in the various organs is characteristic of them, and dependent chiefly upon their embryological development. As soon as a tumour develops in the liver, stomach, or any other organ, we see that their regular formation is replaced by chaotic irregularity. We find, also, in the growing tumour, an extensive new formation of blood-vessels. This is most apparent in the zone of proliferation, which in infiltrating tumours is at the periphery. As the cancerous growth increases in volume, its centre

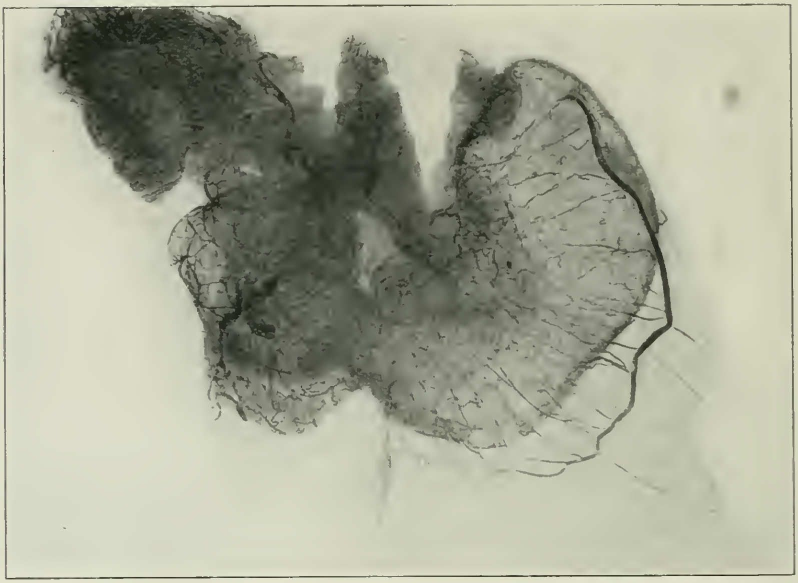

Fig. 1.
Carcinoma of Stomach.

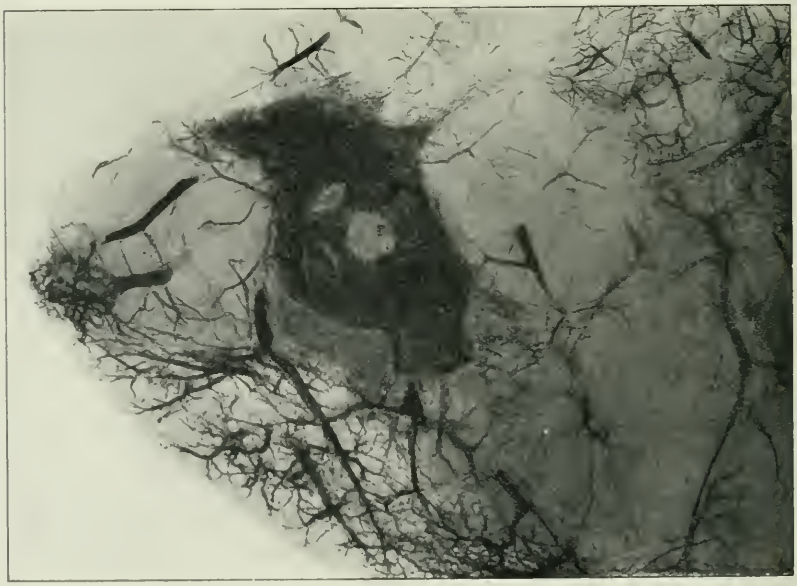

Fig. 2.
Carcinoma of Liver.

becomes necrosed and the newly-formed blood-vessels merely occupy the capsule. Ultimately the blood-vessels seem to disappear altogether, so that in this stage cancer is not rich in blood-vessels, but rather denuded of them. The mass of newly-formed vessels is of small calibre, and their branching is irregular to such an extent that big vessels split up into the smallest of their kind without intermediary types.

In order to amplify the results obtained in man, I have injected spontaneous and experimental growths occurring in mice by means of the following method. After exposing the heart of the anæsthetised animal I punctured the organ and injected Indian ink, avoiding excessive pressure. Indian ink, as the Johns Hopkins School of Anatomy has shown, is a medium which flows easily into the blood-vessels and mixes freely with the blood. By manometrical regulation of pressure we are enabled to modify the result of the injection according to the desired effect. After injection the specimens were fixed and hardened in alcohol. For microscopical purposes transverse and in some cases serial sections of the whole animal were made, which enabled us to gain an exact estimate of the relations existing between the transplanted growth and the surrounding organs. Such specimens likewise enable us to judge the origin of the newly-formed blood-vessels, their number, form and width, as compared with adjacent tissues or organs.

Another set of specimens was cleared after the Schultze method, by placing them first in alcohol, to which a few drops of potassium hydrooxide solution were added, then removing them into glycerinated alcohol, and finally into pure glycerine. Marked differences in the whole arrangement and number of blood-vessels were discovered in the various malignant tumours examined. One common feature was observable in all of them. As soon as infiltrative growth of the transplanted cells sets in, a great commotion is produced in the surrounding system of blood-vessels. The degree of commotion is dependent upon the wealth of preexisting vessels; hence, in strongly vascularised tissue, such as mammary gland or muscle, the vascular irritation is far more pronounced than in subcutaneous tissue which is poor in vessels. This irritation is seen most distinctly in the vessels in front of the growing tumour, and can easily be traced in regions to which the tumour has not yet advanced. The most striking feature of the irritation is the dilation of the affected vessel. It curls itself up into spiral coils and sends forth numerous capillary offshoots towards the invading growth. In carcinoma the newly-formed vessels arrange themselves almost entirely in the peripheral area. As the volume of the growth increases the number of vessels decreases, and eventually, especially in cases of complete necrosis, the vessels totally disappear.

In sarcoma, again, the numerous vessels of new formation are evenly distributed throughout the whole growth, presenting themselves as a delicate, closely-woven network, even in the interior of the tumour. Nowhere are the differences as to vascular supply between carcinoma and sarcoma more apparent than in mixed experimental growths of the carcinoma sarcomatodes type. Even in cleared specimens it is easy to single out the cancerous and sarcomatous spaces by the varied degree of vascular injection.

Wholly different are the appearances in chondroma of the mouse. As Ehrlich has shown, this extraordinary growth presents a blood-red surface on dissection, from which he infers that its cells have distinctly angioblastic properties. I found its capsule rich in vessels, and likewise the derivatives of the capsules, the wide bands of connective tissue, which separate the islands of cartilage. Many of these vessels penetrate into the cartilaginous masses and open out into large vascular spaces, apparently losing all the characteristics of blood-vessels. These vascular spaces disintegrate the cartilage tissue. Numbers of cartilage cells are thus destroyed.

Since these various tumours are produced in the same species of animals by units of implanted cells, the results obtained bring two points into clear prominence. One that has been recently demonstrated by Bashford and others is that the stroma of experimental tumours owes its origin to the inoculated animal, and is not derived from the grafted cells. Another point of greater importance is the fact that the structural qualities of the stroma are determined by the tumour cell.

From what I have said regarding the vascular condition of carcinoma and sarcoma it might appear that there is a fundamental difference between them, inasmuch as the new formation of blood-vessels in sarcoma is so much richer than in carcinoma. This view has been advanced by many of our leading pathologists, who maintain that carcinoma and sarcoma, owing to their genetic affinity to the epithelial and connective tissue cells, react respectively upon the lymphatic and vascular system. Unfortunately, we know next to nothing as to what changes carcinoma induces in the number of lymphatic vessels. All we know is that proliferation of lymphatic glands sets in, in a manner first described by Bayer, and recently confirmed by Ritter, who goes so far as to affirm that the swelling and new formation of lymphatic glands are a typical and early response on the part of the body to the invading cancer cells.

Against the theory of diminished vascular activity in cancer it may be stated that different forms of cancer vary in their wealth of blood-vessels. Bashford has acquainted us with the interesting fact that in his long series of experimental cancers he has observed complete and distinctive difference in the strains. A tumour poor in vessels and much necrosed was transformed into one of an exactly opposite nature. In this connection I may refer to Ehrlich's interesting observation on the transformation of carcinoma into sarcoma in the mouse. As you know, Schmorl was the first to record a similar case in man, one affecting the thyroid gland. Quite recently I had to treat a case of mammary cancer in which complete removal of the breast and the axillary glands on both sides failed to produce a satisfactory result. Numerous secondary growths formed in the front chest wall. After the patient had recovered from a bad attack of pneumonia and extensive suppuration spreading over the front chest wall, the metastatic tumours in the skin broke down and formed polypous, extremely vascular growths, which ultimately led to the patient's decease. On examining these tumours of the skin in their later stages, it was found that all traces of cancer had disappeared, and that fusiform-celled sarcoma had taken its place. On the strength of all this evidence I do not hesitate to declare that the difference in the vascular activity of carcinoma and sarcoma is a quantitative and not a qualitative one. A marked difference exists between the destructive powers of the carcinoma and sarcoma cells, since destruction of blood-vessels is so very much more apparent in cancer than in sarcoma.

I mentioned that the impetus which gives rise to the proliferation of blood-vessels emanates from the invading cell. On the other hand, the study of experimental tumours has proved that the new formation of blood-vessels itself is dependent upon the general powers of the individual to react, as well as upon the physiological condition of blood supply in the invaded tissues. It seems more than probable to me that, in a system weakened by age or pre-existent disease, the vascular reaction induced by the malignant growth is below the normal or fails to take place, thus giving rise to a condition first described by Thiersch as premature senescence of the connective tissue in cancer.

In any case, I regard vascular neoformation as a standard by which we may test the body's power of reacting against malignant tumours. On the other hand, we can measure the virulence *(sit venia verbo)* of the tumour cell by the extent of necrosis found in the growth. I suppose most pathologists of the day are agreed that necrosis is not caused by accidental reasons, such as pressure, malnutrition, and others. Borst has rightly pointed out that in growths highly vascularised necrosis is a common occurrence. I may refer you to the case of chondroma in the mouse, a growth essentially vascular and yet permeated by cell necrosis. Necrosis, as Ritter has put it, is intimately connected with the primary cause of the disease in the same sense that it is in tuberculosis or other infectious diseases. I differ from Ritter in one essential point. He looks upon the border of healthy cancer cells surrounding the necrotic area as a reactive cell proliferation on the part of the body, and the area of necrosis as the tumour *sensu strictiori*. These cells on the necrotic border are, I believe, the militant survivors of the invading group, whereas the body's reaction is marked, as I have tried to show, by the newly-formed blood-vessels and connective tissue. The area of necrosis is, in my opinion, the battlefield on which assailant and defender both perish, for it is not only the tumour cell that is destroyed but the stroma as well.

I have now reached my last point, the consideration of what purpose is served by the new formation of blood-vessels. It would traverse our modern views concerning the functions of the blood to assume that only nutrient and not also defensive material is borne along the newly-created blood-channels. How can we account for the difference of vascularisation in healthy and in diseased blood-vessels on the basis of a nutritive theory? A healthy artery performing vital functions has a minimum of nutritive vessels confined to its outer coat. As soon as disturbance of any kind sets in, blood-vessels spring up and ramify throughout the whole breadth of the vessel's wall. Is it at all probable that the body would produce such a mass of vessels in order merely to feed a circumscribed thickening of the inner coat, frequently the only anatomical lesion definable? Does it not seem far more likely that the newly-formed vessels serve an intensified circulation in the arterial coat, thus guarding it against change from within or without? Here again, as in the case of Ehrlich's side-chains, is an instance of an arrangement in the body originally destined to safeguard one of its organs, but eventually developing into a cause of harm. In this connection I may recall observations of my own, recently confirmed by Schmidt, Lubarsch, and others. Tumour cells constituting a thrombus or embolism may multiply within a blood-vessel and spread along its branches without forming adhesions to the vessel's coat. In case such adhesions form —*i.e.*, as soon as infiltrative growth occurs—the mass of cells is organised in the fashion of the ordinary blood-clot. In such cases it is quite the rule to observe that the cells degenerate, frequently to such an extent that the cancerous nature of the thrombus or embolism is completely obliterated. Only by means of serial sections can we determine the true genesis of the organised thrombus. My own experience has taught me that similar conditions frequently obtain in lymphatic glands, apparently the seat of secondary cancer. Clinically, all the symptoms of metastasis are evident, and yet histological sections of the gland merely reveal inflammatory hypertrophy. This accounts for the great difference of opinion amongst gynæcologists as to the advisability of removing lymphatic glands in cancer of the womb. I do not hesitate to declare that, as in the case of cancerous embolism, careful examination of the glands in serial sections would show that they contain isolated cancer cells, but that the bulk of these cells has undergone degeneration. All these facts prove that the body commands powers of combating cancer and healing it. An overwhelming amount of evidence on this score has been recently brought to light by Lomer. He refers to upwards of two hundred cases of cancer in which the clinical diagnosis was almost invariably confirmed by histological examination, and in which recovery ensued in some without surgical interference, in some after incomplete removal of the growth. In his first series of cases the cure was preceded by constitutional alterations of the blood induced by febrile infections of a general nature, by severe hæmorrhage, by extensive burns, and by blood-poisoning. In the second series the cut surface of the incompletely-removed growth was cauterised by heat or by chemical agents.

Are we justified in doubting the potential efficacy of X-rays and radium in cancer in the face of all that has been recorded in man and animal? If we analyse the anatomical basis of all those cases of cancer in which complete recovery or retarded growth has been achieved spontaneously or by means of mechanical, physical and chemical agents, we always discover the same reaction on the part of the body, namely, the formation of stroma. Of great interest, perhaps also of fundamental importance, is the fact that extensive local hæmorrhage serves as the precursor of this cell proliferation.

From these remarks it might appear that I regard the blood-vessels themselves as agents of defence. On the contrary, I staunchly uphold

Virchow's doctrine, so ably confirmed by Ehrlich's famous researches on the varied powers of oxidation in the body, that the cells are not fed by the blood-vessels, but that the cells feed themselves. Therefore I regard the network of newly-formed blood-vessels merely as useful in producing more active blood circulation. Intensified circulation itself, if I may so call it, is the effect of all those healing powers which I have just referred to, inclusive of inflammatory agents, such as have recently been recommended for the treatment of cancer by Bier and others. The efficacy of this intensified circulation is naturally dependent upon the presence of defensive factors in the blood. It will remain a subject for future research to discover what these defensive substances are, and above all, where they are manufactured. It seems to me that our present clinical and pathological knowledge already enables us to infer that the body's first line of defence is established on the boundary of the invading growth. From this point of view we understand cases like the following, which I believe have come within the notice of every surgeon. Patients suffering from cancer of slow growth and long duration are advised to have it removed instantaneously. The operation is successfully performed, and the healing process is normal. Yet the patient returns within a short time suffering from a recurrence which has grown rapidly and has assumed features of an alarming nature. It appears to me that in such cases the surgeon's knife has done harm. In removing the growth he has destroyed the barrier of defence which the body has carefully raised up during the long period of the tumour's existence. I know full well that many of my surgical colleagues will disagree with me. And yet I feel that the time has come for us to consider whether stereotyped surgical interference is the only remedy of the future for malignant growths. Should we not rather begin to individualise, as we do in every other disease which is brought to us for treatment? But how can we individualise if we pay no attention to the individual characters of the case we treat—if, above all things, we pay no heed to the efforts of the body to ward off the threatening danger? Can we wonder that such contradictory views still exist as to the rational and radical treatment of cancer? All of us know too well what a marked contrast there is concerning operative treatment and its ultimate results in mammary or uterine cancer as advocated by Bryant, Halsted, Wertheimer, Olshausen, and others. Nothing is more harmful to the progress of our science than doctrinary stagnation. We should not pause before the spectre of apparent retrogression. In our attempts to force the stronghold of cancer we fare no better than the engineer whose railroad winds round and round the height he means to pass. And yet how comforting the knowledge that whenever we complete a circuit we have ascended to a higher level of truth.

If I have succeeded in proving to you that it is our duty not only to study the biological problem of the tumour cells, but to gain a deeper insight into the defensive agencies of the body as well, the purpose of this paper has been achieved.

I regard the following as the practical result of my work: A careful study of the vascular conditions prevailing in malignant growths affords an anatomical test of the reactive powers of the body. It will be necessary to collect more extensive evidence on these lines in order to bring it into closer relation with our clinical knowledge of the varied appearance and history displayed by the different forms of cancer affecting the same organ. In future, for example, in the case of the mamma it will not be sufficient for clinical and therapeutical purposes to distinguish between scirrhus, adenoma, &c., from a purely histological point of view as to the arrangement of the tumour cells, their different forms of degeneration, &c. It is equally important to discover their reactive powers on the body as tested by stroma formation. By this means we shall sooner realise my demand for treatment based upon a knowledge of the individual qualities of the growth.

On a future occasion I may be permitted to give you an account of my investigations into the causes of retarded metastases, and into the peculiar vascular conditions prevailing in localities predisposed to malignant growths. Above all things, I hope to be able to report on a first attempt to penetrate into the darkness of physiological conditions existing in malignant growths. I am engaged in research on the varied powers of oxidation and reduction characteristic of the various tumour cells.

I have purposely refrained from comparisons of any kind between malignant growths and infectious diseases, and yet it will hardly have escaped your notice that every point which I have discussed reveals most striking analogies between them. As far as syphilis and tuberculosis are concerned, I have dwelt upon these analogies exhaustively in a paper read before the International Cancer Congress at Heidelberg.

The Value of X-Rays in the Diagnosis of Obscure Abdominal Cases.

By .Professor E. GOLDMANN,

OF FREIBURG.

WITH your kind permission, I mean to give you a short abstract of work which I have pursued during the last few months, with the purpose of applying the X-rays in obscure abdominal disease. At the outset I wish to remark that the X-rays are merely an aid to diagnosis, and only useful when combined with full consideration of the clinical symptoms of the case, and after an exhaustive application of the older methods of examination. I found the X-rays most useful in determining suppuration within the abdominal cavity, in discovering calculus in the appendix, and in the diagnosis of abdominal cancer.

The various forms of suppuration which I examined successfully were subphrenic, prevertebral, and pelvic abscesses. I will confine my remarks to subphrenic abscess. It seems remarkable that, although the literature on this subject has increased to such an extent within recent years, hardly any reference to X-ray diagnosis is to be found. So far as I can see, Albers Schoenberg has been the first and only author who records a successful diagnosis by means of X-rays. In two cases of mine, caused by appendicitis, the ordinary methods of examination failed to prove conclusively that the complication which had arisen was due to subphrenic abscess. On applying the X-rays I found the right pleural cavity unchanged in its transparency. The right half of the diaphragm was pushed upwards. The apex of the deep black convex shadow was of a conical appearance. The diagnosis of subphrenic abscess was corroborated by the operation. In Albers Schoenberg's case the abscess had perforated the diaphragm and ruptured into the pleural cavity. This condition is easily traceable on his plate, which he has kindly placed at my disposal. There are two points of importance to which I must draw your attention. As you know, subphrenic abscess leads to secondary empyema. In my first case this condition had already taken place, and yet the presence of seropurulent fluid in the pleural cavity did not interfere with the distinctness of the subphrenic shadow on the photographic plate. Then, again, the pyothorax subphrenicus, as Leyden has termed it, is often a pyopneumo-thorax. In such cases the gas contained in the abscess naturally modifies its appearance on the skiagraph. In order to avoid errors in diagnosis, due to an abnormal suprahepatic dislocation of the bowel, such as Béclère, of Paris, has reported, it is imperative to take more than one photograph of the case at different times before operation.

Although pathologists, such as Aschoff, have tried to prove that a calculus in the appendix is of no material consequence in the history of appendicitis, yet surgical experience proves that, in 55 per cent. of those cases of so-called chronic appendicitis in which an acute attack occurs, gangrene or perforation of the organ is associated with a calculus. Hence I consider it of great diagnostic value to determine whether the appendix contains a calculus, especially in doubtful cases, where the surgeon is dependent upon the complaints of the patient or the history of a previous attack, ere deciding upon an operation. In the first case of my own, a patient was brought to me with all the symptoms of a retroperitoneal growth. His medical adviser gave me a report of appendicitis, dating several weeks before the patient's reception at the hospital. For diagnostic purposes, I cut down upon what appeared to be a growth, and found in its centre a small cavity containing fæcal pus and a calculus. On re-examining the first skiagraph I had taken of the patient, I found the calculus clearly marked in the sacro-iliac region. On several occasions I have been able to confirm my first experience. Calculus in the appendix, as my plates prove, is easily discernible (fig. 1). Before diagnosing appendicular calculus it is essential to exclude everything which gives rise to the so-called pelvic blotch, a subject so fully discussed at the last International X-Ray Congress. It is necessary, above all things, to differentiate between calculus of the appendix and that of the ureter.

As to abdominal cancer, it seemed a hopeless task to gain knowledge of its existence by means of the X-rays. Even one of our greatest authorities on X-rays, Albers Schoenberg gave it as his opinion that they were of no value in abdominal cases, as the contrast in the absorbent qualities of the abdominal organs is so small. My investigations are founded on the following fact: If we expose a cancerous organ, such as the mammary gland or even the liver, to the X-rays, we find that the growth absorbs the X-rays much more powerfully than the surrounding tissues. The area of cancer appears as a dark space on the plate. Its various roots, and even secondary deposits, are clearly visible (fig. 2).

Were we to make use of this fact without adequate preliminaries in cases of abdominal cancer, our results would be unsatisfactory. In order to achieve the object in view we must endeavour to accentuate the contrast between the growth and its surroundings. This is easily done

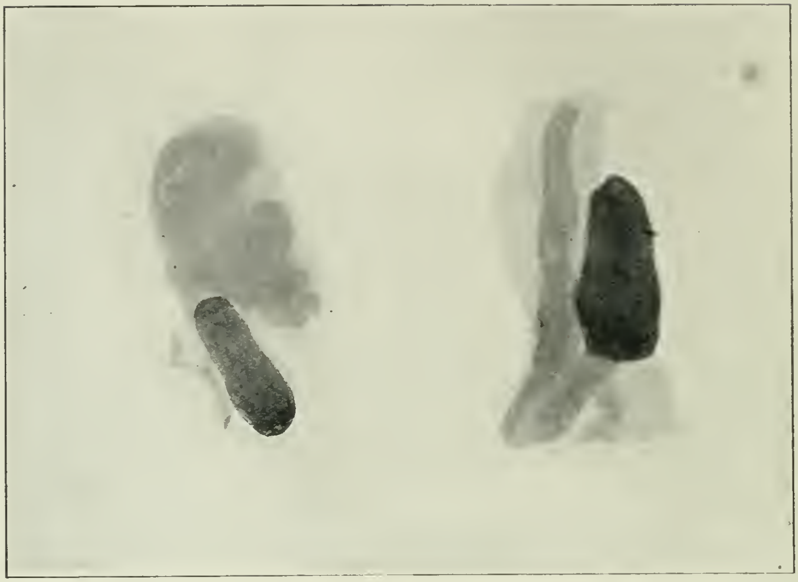

FIG. 1
Calculi in Appendix.

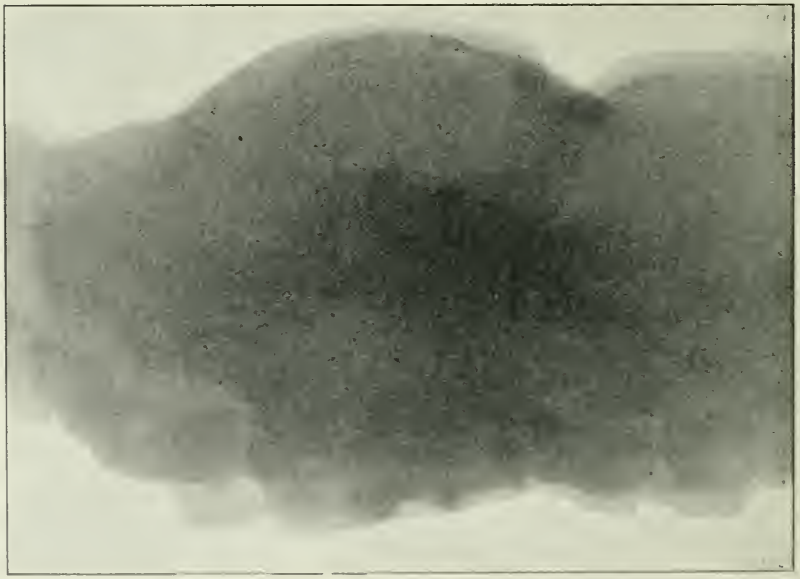

FIG. 2.
Carcinoma Mammæ.

by giving the patient an aperient and subsequently inflating the rectum with air, a procedure borne by the patient without any discomfort. The inflated air rapidly spreads along the gut as far as the ileo-cæcal valve. Under these conditions the abdominal cavity becomes perfectly translucent as regards X-rays, and cancerous growths show most distinctly. In my first case, a patient was brought to me with the symptoms and history of cholelithiasis. On examination I found a tumour in the region of the gall-bladder, pointing conically below the hypochondrium; it was not tender, but very hard. X-rays revealed a dark shadow corresponding to the gall-bladder, and I at once made up my mind that I was dealing with cancer, a fact which the operation, consisting of removal of the gall-bladder, confirmed. The conditions in my second case were very similar to those of the first, the only difference being that the .tumour seemed nodular and of irregular shape. It was clearly definable on the photographic plate. Subsequent operation showed that a growth originating in the duodenum had led to extensive adhesion and cancerous deposits in the gall-bladder. The case was inoperable; death ensued several months after the exploratory incision had been made. Finally I may mention a case of chronic intestinal stricture, which the X-rays proved to be due to a growth occupying the ileo-cæcal valve. I opened the abdominal cavity and found a tumour which both in size and position corresponded exactly to the shadow on the plate.

I am very well aware that my work in its present stage is fragmental and rudimentary, but I hope that greater experience and above all an improved technique, consisting chiefly in a greater sensitiveness of our plates, will enable us to diagnose abdominal cancer by means of X-rays sooner than we have hitherto succeeded in doing. I am making use of the following fact, as yet not sufficiently investigated and utilised: When we expose a photographic plate to light, its sensitiveness does not increase proportionately to the intensity of the reducing rays. Thus the effect produced upon the plate by a slight increase in the brightness of the rays is much stronger than the equivalent degree of increased illumination up to a certain point, which, being different in every plate, should be determined. Since the success of operative treatment of cancer rests upon early diagnosis, and since the possibilities of early diagnosis in abdominal cancer are yet so limited, I have not hesitated in bringing to your notice this fragmentary research, trusting that some of you may feel inclined to join me in improving a method of diagnosis that affords such hopeful prospects for the future.

DISCUSSION.

Sir William Church said he was sure that all present would join in supporting the resolution which he had been asked to bring forward, that a very hearty vote of thanks be accorded to Professor Goldmann for his most interesting and instructive paper and demonstration. Many of the Fellows had seen the microscopical preparations in the adjoining room, which were exceedingly beautiful. They brought before him, as an uninstructed pathologist, very clearly, certain views which he had heard expressed regarding the growth of cancer, or rather of the tissues surrounding cancer. He had been particularly struck with Professor Goldmann's closing remark, that they should not take such a pessimistic view of cancer as had hitherto been taken. It appeared that the natural cessation—he would not say cure—of the growth of cancer occurred more commonly than the bulk of medical men thought. It had been common knowledge to all surgeons that different kinds of cancer, especially carcinomata, did die out. As a physician he had himself seen cases of that kind. It was a valuable fact that the increased attention which had been given to the subject had led to the knowledge that a larger number of such cases spontaneously cease to grow than had been generally supposed. He was sure all present would join in thanking Professor Goldmann for his delightful lecture.

Mr. Henry Morris said he had great pleasure in seconding the vote of thanks which had been proposed by Sir William Church. It had been a privilege and a source of great instruction to him to listen to Professor Goldmann, and he much appreciated being asked to second the vote of thanks. He hoped Professor Goldmann would not assume that the paper had not received due appreciation because only one question had been asked on the subject-matter which had been brought forward. There seemed to him to be three points of instruction and suggestion for research which had been brought forward. Those points were anatomical, pathological, and clinical. The great anatomical point which he understood Professor Goldmann to bring out was the difference of the vasa vasorum of the arteries and the veins, and the intimate communication between the lymphatic circulation and the arterial and venous circulation. And, as Professor Goldmann suggested, that needed further elucidation and enquiry. It must be a matter of the greatest moment, not only with regard to the spread and metastasis of cancer, but with regard to infection generally, if the views which had been outlined were substantiated and verified. The pathological point which he had enforced was that more importance should be attached to the physiological resisting power of the body against the cancer cell. For many years past there had been rather a tendency to concentrate attention upon the cancer cell and its various changes and phases, and whether the cancer cell was the site of a micro-organism which was the cause of the disease. Perhaps that had been too much emphasised in the past; what was wanted was not so much the persistent and uninterrupted use of the microscope, but also the field-glass, which would permit of an extensive view, and at the same time focus the different points of a large outlook. That phase of the address pointed to the necessity of our being on the look-out for the effects of various agents in strengthening the physiological power of the system against the growth of cancer; and he was glad that Professor Goldmann had said what he had in regard to radium and the other measures which had been employed, which he hoped would not be put on one side with any undue haste and condemned without further research. The same remark applied also to the toxin treatment. He had been very glad to hear what had been said on the clinical point, namely, as to the selection of cases of cancer for surgical interference. Mr. Morris was sure that it should not be taken as a routine procedure that because a person had cancer which was capable of removal, therefore it ought to be removed. It had been his great experience in life to have had frequent opportunities of following the surgical practice of Campbell de Morgan, Charles Moore, and Hulke, and he was familiar with the kind of case referred to, the small hard scirrhus in an old and thin person, which had existed for many years. The sound advice given in those cases by the above-mentioned surgeons was to leave them alone, because the experience of these authorities was that after removal there commonly occurred a very rapid return of the growth. That point alone in the clinical aspect was one of very great use to all.

The vote of thanks was carried by acclamation.

Dr. F. J. Smith said he could not pretend to discuss the paper; the whole matter of it was practically in the hands of the surgeons. But he would like to ask a question, as a physician, on an expression used towards the close of the address. It was that in certain cases of carcinoma the surgeon, by excising the growth freely, produced rather more harm than good, because he was cutting off the protective barrier against the spread of the disease at the same time as he was removing the disease. Every one—physicians as well as surgeons—came across cases in which the question was asked as to whether it was cancer or not, and whether operation was desirable; and he would be glad if Professor Goldmann would indicate the class of case in which no operation should be performed. Of course, each could use his own common sense as to whether an operation should be attempted when a particular organ was involved, but the address led him to believe that some line of demarcation had been found between the cases which ought to be operated on and those which ought not. If so, he would be very glad to hear it.

Professor Goldmann, having acknowledged the vote of thanks, said, in reply to the question asked by Dr. F. J. Smith, that he could not do better than refer him to the great work on the subject by von Bergmann in connection with scirrhus of the breast. The experience had been the same in France, America, and other countries. Von Bergmann's results were no worse than those which Wertheim had published, following the so-called radical operation of excision and removal of glands. In Professor Goldmann's opinion, many cases of cancer of the uterus had been lost by attempts on the part of surgeons to excise all the glands in the vicinity of the disease. Yet frequently those glands, examined after the death of the patient, did not contain any cancer cells at all, or, if any, only in such isolated small numbers that the body might be able to deal with them. He was not so pleased with the results of his own experience as were other surgeons. He did not think it was sufficient simply to disbelieve the statement of some operators that in excising the mamma one need not remove the axillary glands, divide the clavicle, and even clear away the supra-clavicular glands. It was impossible for him to say where surgical interference should stop; his own aim had been to draw attention to the matter: there was need for the collection of much more evidence on the point. Sufficient was not yet known about the subject. One only heard or read reports from one side in the case of men who did radical operations, and on the other side from men who did not. Thus there were two conflicting opinions, and there was not yet any solution. The only way to solve the problem was to approach it in the way he had indicated, by trying to find out whether, in the one case, the barrier of defence was destroyed by operation, or whether, in the other, it was better to remove the growth. It was a question which pathologists alone could not decide; pathologists should work hand in hand with surgeons towards the elucidation of the problem.

Surgical Section.

November 12, 1907.

Mr. J. Warrington Haward, President of the Section, in the Chair.

An Analysis of 274 Additional Cases of Removal of Goitre by Operation.

By James Berry, F.R.C.S.

Mr. President, Ladies and Gentlemen,—In previous communications I have published the whole of my operations (126 in number) for the removal of goitre, both innocent and malignant, up to February, 1901.[1] The present series of 274 cases is a direct continuation of the former series, and brings the total number of operations up to 400. It embraces every operation for the removal of goitre of whatever kind performed by me during the six and a half years ending September 16, 1907. The 274 operations now recorded were performed upon 268 patients, of whom 32 were men or boys and 236 women or girls. One man was operated upon five times for recurrent papilliferous tumour, and two women twice, for papilliferous tumour and recurrent parenchymatous goitre, respectively. Seventy-five of the operations were performed in private practice, and 199 in hospital practice. The ages of the patients varied from 12 years (No. 144) to 73 years (No. 338) and are shown in the accompanying table :—

Table I.—Sex and Age.

Sex ...	Males 36 operations on 32 patients		274 operations on 268 patients.
	Females 238 ,, ,, 236 ,,		

Years of age :—

	Under 15	15–19	20–29	30–39	40–49	50–59	60–69	over 70	Total
Operations ...	6	19	61	92	59	29	7	1	274
Patients ...	6	19	60	91	59	25	7	1	268

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[1] In a later paper (*Lancet*, 1902, i., p. 1227) I have given a mere summary of results up to case 155, at the end of February, 1902. Two of the cases described in that paper (Nos. 135 and 136) come within the present series.

It will be noticed that no less than 95 of the operations were performed upon people of the age of 40 and upwards; 8 of these patients had reached the age of 60. Many of the operations were very extensive and formidable.

With regard to the provenance of the patients, about three-quarters of the whole number have been sent to me from various parts of England outside London; about a quarter come from London itself, but of these the majority had previously lived in the country. A few came from Wales, Ireland, and various countries abroad. The greater frequency of goitre in country districts is, of course, a marked feature of the disease, and the explanation of it is to be found, I believe, in the fact that those who live in large towns drink, as a rule, water from good public supplies. Those who live in country places often obtain their drinking water from open streams, wells, and other sources of supply far more likely to contain the goitre-producing poison, whatever that may be. At the same time it must be borne in mind that, in a considerable minority of cases, especially of encapsuled tumours, the nature of the drinking water has probably little or nothing to do with the causation of the goitre. Nearly all the patients were sent to me by doctors, and had undergone, in most cases, treatment of various kinds for longer or shorter periods.

This paper deals only with those cases that, for one reason or other, I have thought fit to treat by the operation of removal. It need hardly be said that the great majority of cases of goitre require no operation at all. Most cases of parenchymatous goitre are better treated by internal remedies; many other goitres, although not absolutely curable by medicine, cause little or no trouble, and require no active treatment at all. It is only in a minority of cases that operative interference is demanded, or even permissible.

Coming now to the *reasons for operation* in these cases, they are to be seen in Table II.

TABLE II.—CHIEF REASON FOR OPERATION.

	Cases
Dyspnœa	in 177
Deformity	,, 33
Discomfort or deformity, mostly with minor degrees of dyspnœa ...	,, 47
Malignancy, papilliferous tumour, &c.	,, 10
Suspected malignancy	,, 3
Dysphagia	,, 1
Increasing size	,, 3
	274

By far the most important reason for operating upon a goitre is dyspnœa; in 177 cases, approximately two-thirds of the total number, this was the principal reason that led to the operation. With few exceptions dyspnœa, which will not yield to milder remedies, is the only reason that should lead a surgeon to urge an operation upon a goitrous patient. And let me here say that no degree of dyspnœa that I am acquainted with is too severe to permit of an operation for its relief. I have occasionally been told by medical men that so-and-so has a goitre which causes so much difficulty in breathing that no operation is to be thought of. But it is just in such cases that operation can do most good, and should be most strongly urged. I have never, on the ground of the severity of the dyspnœa, refused to operate upon any case of non-malignant goitre, but I have, in scores of cases of parenchymatous goitre, declined to operate on the ground that there was not sufficient dyspnœa to justify operation, or that the goitre could be more satisfactorily treated by the administration of iodine, iodide of potassium, thyroid extract, arsenic, or some other internal remedy.

I may here be permitted to express a strong opinion that the dyspnœa of innocent goitre is always caused by direct pressure upon the trachea. It has little or nothing to do with irritation of the recurrent nerves, an obsolete notion that should be given up. Further, the trachea is displaced or compressed by goitre according to definite and fixed rules, so that it is nearly always possible by careful consideration of the nature, shape, and position of the goitre, to predict the position and shape of the compressed trachea. Thus a uniform enlargement of the thyroid gland produces a bilateral compression. A strictly unilateral goitre bends the trachea over to the opposite side, and flattens it obliquely on the side of the tumour. Bilateral and asymmetrical goitres cause various forms of oblique flattening and often some rotation. A knowledge of the probable shape and position of the trachea is often important to the surgeon when sudden dyspnœa threatens life in the course of an operation. By pulling upon the tumour in this or that direction he can relieve the pressure. Perhaps more important is it to remember that he must be careful not to exert pressure or traction in such directions as to increase still more the compression of an already compressed trachea.

The next on my list of causes is deformity, which alone was the cause of operation in thirty-three cases, while in forty-seven others deformity, associated with minor degrees of dyspnœa, led to the operation. As a rule, it is only in the case of young people, and for encapsuled tumours, that operation should be undertaken on the ground of deformity alone. In such cases it may fairly be stated that the operation is as nearly devoid of risk as any operation can be, provided that it be performed by practised hands, and with due attention to details. In three cases only has operation been undertaken for patients over 40 on the ground of deformity alone. Unilateral tumours, situated well above the sternum, are those which usually produce the greatest amount of disfigurement, and these are precisely the cases that can be operated upon with the greatest ease and safety. Some of the largest and most prominent goitres that I have had to deal with have been among the easiest of my operations.

Dysphagia is in itself rarely a prominent symptom of an innocent goitre, although its early occurrence is frequently a very valuable indication of malignancy. Dysphagia may, however, be a marked feature of those somewhat rare cases in which a small cyst or adenoma develops at the inner and back part of a lateral lobe. Being bound down by the layer of cervical fascia that passes from the back of the gland to the posterior aspect of the pharynx or œsophagus, and being in close proximity to the recurrent laryngeal nerve and the posterior ends of the tracheal arches, a tumour in this region, even when quite small, no larger than a hazel nut, may cause rather severe symptoms of dysphagia, dyspnœa, and general discomfort. Cases 282 and 378 may be mentioned as examples.

Minor degrees of dysphonia, or even aphonia, occasionally occur in connection with innocent goitre. When we consider the close anatomical relation which the recurrent laryngeal nerve bears to the thyroid gland, it is, I think, somewhat remarkable that innocent thyroid tumours so seldom cause symptoms referable to this nerve. Tumours originating in the inner and back part of the gland occasionally seem to stretch, and thus paralyse the nerve. Inflammation of a thyroid tumour will occasionally produce the same effect. Case 351 was that of a lady, aged 54 years, who had had for many years a solid adenoma as large as a hen's egg in the right thyroid lobe. For eight years she had had dysphonia, and the right recurrent nerve was found to be completely paralysed. Resection-enucleation of the tumour completely relieved the rather severe dyspnœa from which she was also suffering, but the long paralysed nerve had not regained its function when I last saw the patient, seven months after the operation, nor is it likely that it ever will do so. On the other hand, when the dysphonia is of recent duration, removal of the tumour will probably restore the nerve to its function, a matter of some importance to singers. Case 323 was that of a public singer who found that she was losing her voice. Resection-enucleation of a mainly substernal parenchymatous goitre, weighing 5 ozs., completely cured both dyspnœa and dysphonia, and enabled her to resume her profession with success. Case 387 was that of a nurse, aged 30 years, with almost complete aphonia, due to the pressure of a soft solid adenoma of the size of a small orange. Resection-enucleation cured her dysphonia and restored her voice. In neither of these two cases, however, was there any definite paralysis of the nerve. Several others of my patients have also told me that removal of the goitre had enabled them to resume singing.[1]

The *nature of the goitre* in the various cases is indicated in Table III.

TABLE III.—NATURE OF THE GOITRE.

	Cases	Cases
Encapsuled—		
Solid adenoma	in 106	
Cystic adenoma	,, 94	
Pure cyst	,, 2	
Hydatid cyst	,, 1	
	——	203
Non-encapsuled—		
Parenchymatous	,, 26	
Adeno-parenchymatous	,, 28	
Papilliferous	,, 10	
Malignant	,, 7	
	——	71
		274

For purpose of operative surgery it is extremely important to differentiate between cases of encapsuled tumour (cystic or solid) and those cases in which there is a more or less general enlargement of the whole gland (the parenchymatous or adeno-parenchymatous goitres). The former, as a rule, are best dealt with by some kind of intraglandular enucleation; the latter require the extra capsular operation of extirpation, or some modification of this.

The terms *solid adenoma* and *pure cyst* explain themselves. It will be noticed that in the whole series there are but two cases of pure cyst. The 94 cases of cystic adenoma, however, include a good many cases that would probably by many surgeons be termed simple cysts. I have shown elsewhere [2] that nearly all thyroid cysts of considerable size begin life as solid adenomata. These tend to break down and liquefy, usually in the centre, and as this process extends the tumour may eventually become a cyst with mere traces of the original adenoma adhering to the inner wall of what is now practically a cyst. The 94 cystic adenomata in my list include all stages of this degeneration, from the nearly solid adenoma with but little cystic degeneration, up to nearly pure cysts. A pure cyst is one in which, on careful examination, no trace of the adenomatous tissue can be found adhering to its inner wall. It has a smooth inner surface like that of a hydrocele. In many of the solid adenomata the converse process of fibrosis may be observed, beginning often in the centre, where a star-shaped mass of dense fibrous tissue is to be seen (see Case 284). The average age of the 107 patients with solid adenomata was 37·89 years. The average weight of the tumour in the 64 cases in which the weight was recorded was 5·42 ounces.

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[1] Other cases of this class are 168, 177, 185, 216, 253, 271, 351, and 383.

[2] *Trans. Path. Soc. Lond.*, 1896, xlvi., p. 234: "On the Transformation of solid Thyroid Adenomata into Cysts."

Hæmorrhage into thyroid cysts and into the softer forms of solid adenoma is extremely common, and is illustrated by many of my cases. Hæmorrhage into such a tumour is probably the commonest cause of sudden dyspnœa in a unilateral goitre. If the adenoma or cyst is already the cause of considerable dyspnœa, a sudden increase in size from hæmorrhage may easily lead to most alarming and, in some cases, even rapidly fatal dyspnœa. Not many years ago a young woman, who was attending the out-patient department of one of the large London hospitals for a unilateral thyroid swelling, actually died on the doorstep of the hospital from sudden dyspnœa. The post-mortem examination showed an extensive hæmorrhage into an encapsuled tumour. A striking, but fortunately less serious instance, of sudden hæmorrhage causing dyspnœa, is afforded by Case 294. A woman, aged 22, who was not aware of having anything wrong with her neck, was suddenly seized at midnight with alarming dyspnœa, for which she was seen shortly afterwards by Dr. Lambert Lack. He found a small substernal goitre, which I enucleated a few hours later. The small tumour which was pulled up from behind the sternum is on the table before you, and it will be seen that it consists largely of extravasated blood. I find that in 17 of my cases it is definitely noted that blood extravasation was present, but the condition is such a common one that I have no doubt there were many other cases in which no special note was made of the occurrence.

The term *parenchymatous* goitre, although not free from objection, has been retained in this paper to include all forms of uniform and general enlargement of the gland, except that of Graves's disease. It is the common form of goitre in young people. The average age of the patient in twenty-six operations of this class was only 21 years.[1]

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[1] Excluding rather exceptional cases of women, aged 30 years or more, the average age of the remainder was 18·38 years (of nine males, 18·44; of twelve females, 18·33 years). The average weight of the goitre removed in twenty-five of these cases was 7·83 ozs., just under half a pound.

Adeno-parenchymatous are those goitres, originally purely parenchymatous, in which one or more adenomata are present. The tumours are embedded in the goitre, like pebbles in pudding-stone. As the adeno-parenchymatous is a later stage of the disease than the parenchymatous, the average age of twenty-eight patients in my list with this variety of goitre was 33·33 years. The average weight of the tumour (recorded in twenty-one of these cases) was 5·86 ounces.

The situation of the goitre, with regard to the sternum and thorax, is a matter of much importance. Goitres situated well above the sternum are not, as a rule, dangerous, unless they are bilateral, or of very large size, and even then but rarely. But the nearer the goitre approaches to the thorax, the more dangerous is the dyspnœa produced thereby, since a sudden swelling of the tumour, or a sudden displacement of it in a confined space behind the sternum, may cause very severe pressure upon the trachea. The most dangerous of all goitres is the rapidly growing bilateral goitre of young people, situated quite low down behind the sternum. In such cases the trachea is apt to be greatly flattened, and it is soft and yielding, and therefore easily compressed to a further and fatal degree. Such cases, if attended with marked stridor, and not yielding very quickly to medicinal measures, should, in my opinion, always be submitted to operation. Unilateral goitres, in this low situation, occur as a rule in older subjects, in whom the trachea is more resistent and less liable to collapse. Danger of sudden death in these cases arises mainly from hæmorrhage into the tumour, or from the tumour being suddenly sucked downwards by inspiration into the upper opening of the thorax. Marked stridor, with a substernal or intrathoracic goitre, is nearly always an indication for operative interference. Among the most satisfactory cases in thyroid surgery are those extreme cases in which the main bulk of the goitre is actually engaged in the thorax itself—the intrathoracic goitres.

The upper opening of the thorax, a hard bony ring formed by vertebral column, first ribs and sternum, permits of no outward expansion of the goitre engaged within it. All the pressure of an enlarging goitre is, therefore, directed against the trachea and other structures which pass through this small oval ring. It may here be remarked that it is not uncommon for an elderly patient with a unilateral goitre to give the history that her goitre has been gradually getting smaller while her dyspnœa (or asthma, as she often calls it) has been getting worse. This means that the tumour is descending towards the thorax. Case 243 is an excellent illustration of this.

Of non-malignant *intrathoracic* goitres there are seven in my list. Of these the youngest (Case 231), a woman aged 29 years, had only moderately severe dyspnœa, as the comparatively small intrathoracic goitre had not yet grown sufficiently to exert a dangerous degree of tracheal pressure. All the others (Cases 182, 231, 232, 243, 248, 254, 365), whose ages ranged from 37 and 38 years up to 56 and 60 years, had high degrees of dyspnœa, and the relief afforded by operation was correspondingly great. All made excellent recoveries. I have not included among the intrathoracic goitres those in which the tumour was merely substernal, that is, engaged between the sternum and vertebral column, although many of these produced also severe dyspnœa; nor have I included any of those cases, common enough, in which the tip of the inferior horn (as in 312 for instance), although extending actually into the thorax, was not of sufficient size to cause injurious pressure there. One of these cases of intrathoracic goitre (No. 248), was of so remarkable a nature as to demand a more detailed description.

The patient, a man aged 37 years, was sent to me by Dr. P. F. Barton, of Wimbledon, on account of gradually increasing dyspnœa of several years duration, and a thyroid swelling at the root of the neck. During the last three years he had had seven or eight attacks of more severe dyspnœa. Medicinal treatment had always succeeded in reducing the size of the swelling, but never caused its complete disappearance. There was a general fulness of the root of the neck, and the top of a swelling, evidently thyroid, could be felt rising up behind the sternum. There was great dilatation of all the superficial veins about the root of the neck, and there was stridor on deep breathing and coughing. With the laryngoscope the trachea was seen to be displaced slightly to the left. The diagnosis made was "probably encapsuled tumour of right lobe, substernal." Operation was performed on May 3rd, 1904, when it was found that both inferior horns extended deeply into the thorax. A forefinger, passed as far as it would go into the thorax on the right side, felt a large rounded swelling well below the level of the first rib, but the lower border of this swelling could not be felt. An attempt was made to pull the tumour up out of the thorax, but failed completely, as the tumour was too big and too solid to pass through the narrow bony opening. Attention was, therefore, directed to the left inferior horn, where a similar but somewhat smaller tumour lay in the corresponding situation also within the thorax. This tumour was fortunately partly cystic, and after letting out some two ounces of fluid from it, its size was diminished sufficiently to enable me to draw it up out of the thorax, and remove it by enucleation. Returning to the first tumour, some of its solid contents were scooped out with the finger, and a calcified portion could be felt. This was seized from within by a long pair of forceps, and the tumour, now much diminished in size, was finally dragged up and enucleated. The patient made a rapid and excellent recovery, being up on the third day, out of doors on the sixth, and on the ninth he returned to his home with a superficial granulating wound as large as a sixpence, which soon healed. I hear from Dr. Barton that he is perfectly well, and has had no return of his dyspnœa.

Coming now to the *nature of the operations*, these fall naturally into two main classes, as will be seen in Table IV.

Table IV.—Nature of the Operation.

Enucleation.	Innocent goitre.	Malignant goitre.
Enucleation (simple)	107 ...	1
Resection-enucleation	92 ...	0
Evidement	3 ...	0
Enucleation and evidement	2 ...	0
Resection-enucleation and enucleation	4 ...	0
	208 (with 1 death)	1 (with 1 death)
Extirpation.		
Extirpation (simple)	19 ...	6
Resection-extirpation	33 ...	0
Resection	1 ...	0
Resection and extirpation	1 ...	0
Extirpation and enucleation	2 ...	0
Resection-extirpation and enucleation	1 ...	0
Resection-extirpation and resection-enucleation	2 ...	0
	59 (with 2 deaths)	6 (with 2 deaths)
Total Innocent goitres ...	267 (with 3 deaths)	274.
Total Malignant ,, ...	7 (,, 3 ,,)	

When an encapsuled tumour is present, the operation of choice is intraglandular *enucleation*. When the goitre occurs in the form of a more or less general enlargement, then the extraglandular operation of extirpation must be chosen. When numerous small encapsuled tumours are present in an otherwise parenchymatous goitre, extirpation is usually to be preferred. The largest number of encapsuled tumours that I have enucleated at any one operation has been six (Case 337).

It is important to bear in mind that an encapsuled tumour however large, however prominent, and however superficial it may appear to be, never makes its way through its covering of gland tissue. It is always covered by an expanded and more or less thinned and atrophied layer of gland tissue. This layer, which is often no thicker than tissue paper, and may closely resemble a layer of fascia, must be cut through before enucleation can be performed. The region of safety is inside this glandular capsule, not outside it. When this layer of gland tissue is very thin or adherent, as it often is, to the front of the tumour, it may be difficult to separate it from the surface of the tumour itself. For such cases it may be better to incise the capsule at the upper or lower border of the tumour, where the gland tissue is thicker, and more easily separated from the underlying tumour. The tumour itself can usually be readily recognised by its colour, which is almost always quite different from the maroon red colour of the comparatively healthy gland.

A very important modification of enucleation, and one which I now almost always employ for encapsuled tumours, especially when they are large, is *resection-enucleation*. After exposure of a lateral lobe it is dislocated forwards (whenever possible) and an incision made through the glandular capsule till the tumour is reached. This is then enucleated on its inner and posterior aspects, and the glandular layer is again divided. The tumour is then removed together with a large part of the thinned, atrophied, and functionally useless layer of gland tissue covering it. By this means the area of enucleation from which bleeding takes place can be greatly diminished, a point of much importance. Care must be taken in incising the gland to avoid cutting the recurrent laryngeal nerve, which may be displaced laterally to a certain extent. After removal of the tumour the cut edges of the gland are usually drawn together and united by sutures so as to diminish still further the area of wounded gland-tissue.

Evidement is a proceeding of but little importance. It consists in cutting the tumour across and scraping out its contents from the inside. It is chiefly applicable to soft, solid tumours with very thin walls, which may be difficult to enucleate satisfactorily. It is occasionally required for fixed adherent tumours which cannot be lifted out of the glandular bed in which they lie.

It is scarcely necessary, I think, to describe so well known an operation as that of *extirpation*. I would merely say that it is important in this operation that all vessels of appreciable size should be tied or clamped before they are cut, and before any attempt is made to remove any part of the gland. Especially important is it to tie securely the veins at the lower horn of the gland. A cut and unsecured vein in this region will retract deeply into the cellular tissue at the root of the neck, and it may be exceedingly difficult to find it again. Moreover, blood extravasated into the cellular tissue of the neck or mediastinum is prejudicial to the healing of the wound.

Throughout this operation the surgeon should, in my opinion, proceed deliberately and carefully, and see exactly what he is doing at each stage of the proceeding. I may add that the superior thyroid artery and vein should be tied at an early stage of the operation. With regard to the inferior thyroid artery, I have almost wholly abandoned the practice of tying the main trunk of this artery in its continuity, and prefer to clamp and tie the branches after its division, and beyond the point where it is crossed by, or crosses, the recurrent nerve.

I need hardly say that *resection-extirpation* bears the same relation to extirpation that resection-enucleation does to enucleation. Instead of dissecting the inner and back parts of the gland away from the trachea, œsophagus and recurrent nerve, the knife is carried boldly through the gland, and this portion of it is left behind. This proceeding has the double advantage not only of avoiding the dangerous region of the recurrent nerve, but also of leaving behind enough to carry on the function of the gland on that side, and also to fill up the unsightly hollow that would otherwise be left in this part of the patient's neck.

Coming now to the consideration of certain points common to all thyroid operations, the important question of *anæsthesia* may first be discussed. I venture to think that if due care be exercised in the administration, a general anæsthetic may be given in most cases, exceptions being made in certain cases where high degrees of tracheal stenosis and cyanosis are present. In five cases only of the present series was a general anæsthetic dispensed with. Whenever possible, the services of a skilled anæsthetist should be obtained for all but the simplest of thyroid operations. The anæsthetic should always be administered in the room in which the operation is to be done, and not in any side room. The preparatory dressing on the neck should be loosened and the operator be quite ready to begin the operation instantly, should sudden respiratory difficulties require him to do so. Immediate and rapid operative interference is occasionally required even in cases where no unusual difficulty has been anticipated. (See Case 144.)

Chloroform has been the anæsthetic employed in nearly all cases, sometimes combined with ether. I am aware that some operators prefer ether administered by the open method and there is much to be said for the practice. The nature of the anæsthetic is, however, of less importance than the method of administration. Deep anæsthesia is dangerous. It is enough if the patient is insensible to pain, and especially to the pain of the first incision. Anæsthesia should be induced very slowly and carefully, and a minimum of anæsthetic should be employed. The various anæsthetists who have given me the benefit of their services in these operations, have often told me that not more than 2 or 3 drachms of chloroform have been employed in the whole course of the operation. Towards the end of the operation, and before the wound is closed, the anæsthesia should be so light that, at a given signal, the patient can be made to retch and strain, and thus test the efficacy of the hæmostasis. In the course of an extensive operation a small vein may easily escape ligature, and in the movements of vomiting that occur after the patient has returned to bed, sufficient hæmorrhage may take place into the wound to endanger the rapid healing of the wound, if not actually the life of the patient. The main danger of a large thyroid operation is *post-operative venous hæmorrhage.* If hæmorrhage is going to occur, it is best that it should take place while the wound is still open, and while it can easily be dealt with.

The *position of the patient* should be that of semi-recumbency, with the head thrown back as far as the anæsthetist will permit. Occasionally the operation has had to be performed with the patient sitting nearly bolt upright, *e.g.*, No. 186. A curved transverse *incision*, very low down in the neck, in the situation usually occupied by a necklace, is the incision that I now almost invariably employ. (See Table IV. A.).

TABLE IV. A.—SKIN INCISIONS.

Oblique	31
Curved transverse	240
Vertical	1
Angular	1
Y-shaped	1
	274

Of the last 180 only 4 were oblique; 1 was Y-shaped and 175 were transverse.

The results as regards the scar are infinitely better than when any other form of incision is employed. This is a matter of much importance to ladies who desire to wear a low dress. Skin and platysma are dissected up, and some division of the infra-hyoid muscles is generally made high up in the neck. The musculo-fascial layer is then split in the middle line nearly down to the sternum, so that the drainage tube may afterwards lie in the correct position. The infra-hyoid muscles are sutured towards the close of the operation. *It is important that the wound should be kept covered up with layers of wet gauze as far as possible throughout the operation,* to diminish risk of wound infection. Rubber gloves should always be worn by the operator and by all who take part in the operation. Very fine silk, boiled immediately before use, has been used for all ligatures and sutures, except those of the skin.

As is shown in Table IV. B., I have come to employ drainage more and more frequently. A study of the temperature charts of the whole series lying on the table before you will, I think, bear out my contention that the wounds heal more quickly and more satisfactorily if drainage be employed for a short time. After all large goitre operations there is for some hours a considerable exudation of blood-stained serum and colloid. It is better that this should drain away rather than be left in the wound to be absorbed, or to be a source of subsequent trouble. On the other hand, prolonged drainage is usually unnecessary and harmful. My usual practice now, in all cases except trivial ones, is to drain with a single rubber tube (two sometimes, if the operation has been bilateral) placed usually in the episternal notch. This is almost invariably removed at the end of from 18 to 24 hours and a very slender strip of sterile gauze is usually inserted for another 24 hours. Occasionally, when there is much secretion from the wound, the tube or gauze may have to be kept in a day or two longer.

TABLE IV. B.—DRAINAGE OF WOUNDS IN THE LAST 300 OPERATIONS.

Nos.	Drainage	No drainage	Not noted
101–200	69	31	0
201–300	86	13	1
301–400	94	6	0
Totals	249	50	1

In most cases the lowest part of the wound will be found to lie, to some extent, below the level of the upper border of the clavicle or sternum, and in cases of substernal or intrathoracic goitre, a very large space may be situated here which is difficult to drain. It is remarkable, however, how quickly such cavities contract and fill up if they be drained only for a day or two. The most troublesome of such cases are those of old people, in whom the parts are more rigid, and in whom fibrosis and calcification of the remaining parts of the thyroid may hinder due contraction of the wound. If drainage seem difficult, much may be done by postural methods. Sitting up in bed, or lying on one or other side, may favour drainage. Frequently, on the day after the operation, the patient is laid flat in bed, and then rolled over on to the face for a few minutes, two or three times a day. In cases where more prolonged drainage is necessary, the frequent assumption of the genu-pectoral position is a great help. After operation, patients sit up in bed from the beginning, except in cases where the operation has been unusually severe, or the patient is old, and likely to be faint. In such cases the patient may lie on the side opposite to that from which the goitre has been removed. Patients usually get out of bed for an hour or two on the evening of the day following the operation, and most patients are quite well by the middle of the second week, or sooner. Stitches are removed on the fourth day. The cut edges of the platysma are united at the time of operation by buried sutures.

Table V. shows the results of the operations as regards the healing of the wounds:—

V.—HEALING OF WOUND.

	In 267 non-malignant cases		In 7 malignant cases
(1) Immediate healing by primary union	232	...	3
(2) Primary union, except in track of drain; healing in from 10 days to 3 weeks	11	...	1
(3) Secondary union after drainage and gauze packing; healing in from 12 days to 5 weeks ... [4 of these intra-thoracic (cases 182, 243, 248, 254), 1 operation during insensibility from asphyxia (case 136).]	7	...	–
(4) Apparent primary union; late mild suppuration after leaving hospital, home, &c.; eventual complete healing	8	...	–
(5) Mild suppuration, chiefly superficial, stitch abscesses, &c., in hospital or home; healing in from a few days to 2 months	5	...	–
(6) Rather profuse suppuration and sinus for several weeks, then complete healing (case 272) ...	1	...	–
(7) Died	3	...	3
	267		7

Complications of any kind are rare: they have all been recorded in the accompanying statistical tables. The only serious complication occurring during the operation was an unfortunate accidental wound of the crico-thyroid membrane (Case 159). This took place in the separation of the larynx from a large tri-lobed parenchymatous goitre in which it was buried. A septic element was thus introduced into the extensive wound, necessitating open treatment, and the patient died of pleurisy on the eleventh day. The portion of the goitre removed weighed 22 ounces. One patient (Case 244), a stout, elderly gentleman with a partly substernal goitre, and considerable dyspnœa, had some bronchitis for a few days after the operation, and for five days his temperature was over 100° F. He then made a rapid and excellent recovery, and left the nursing home on the twelfth day after the operation. In one case (Case 358) recurrent venous hæmorrhage, after removal of a deeply seated solid adenoma, made it necessary to open up the wound nineteen hours after operation, and to remove a handful of clot. The wound was packed with gauze, and the patient made a good recovery; the temperature never rose above 100·2° at any time. In one other case (Case 209) some hæmorrhage from a subcutaneous vein occurred when the drainage tube was removed on the day after the operation. The wound was partly reopened, and the vein tied without any difficulty. In three other cases a little blood collected in the wound and delayed its healing a few days. In one case the same cause led to some suppuration, and a sinus lasting for several weeks. None of these last six cases were seriously ill at any time. In two cases, mild inflammation of the wound led to its being reopened and drained on the second and seventh days respectively. In two cases only was any damage inflicted on a recurrent laryngeal nerve. In one of these, part of a recurrent nerve was intentionally removed, being involved in a hard papilliferous tumour, believed to be malignant; in the other mild suppuration, after enucleation of a cystic adenoma, was found five months later to have caused paralysis of one cord. The larynx is examined in all cases, both before and after operation. One patient was the subject of acromegaly at the time of operation, but this made no difference to the satisfactory healing of the wound.

Neither cachexia strumipriva (myxœdema), nor tetany, have occurred in any case. All cases are now asked to report themselves periodically, and an attempt has been made to ascertain the subsequent history of each case. Most of the earlier cases were examined and reported on in 1906, and the later ones at various times during the present year. The date at which each patient was last seen is recorded in the tables. Fifteen patients I have been unable to trace; in all of them the wounds healed by first intention, and I have no reason to suppose that any of them are at the present time in any other condition than that of perfect health.

Among the 267 non-malignant cases there were three deaths; of these one has already been narrated, the other two were from cardiac failure shortly after the operation. One was No. 288, a boy aged 17, admitted with a large bilateral parenchymatous goitre and chronic severe dyspnœa. More than a year previously, his doctor, Dr. Kendall, of Chiddingfold, had urged him to have the goitre removed. But he had delayed and delayed until he had reached the last stage of chronic dyspnœa, and his heart had begun to fail. At the operation, which was done under local anæsthesia, the tracheal walls were found to be almost in contact, only a narrow chink at the anterior part permitting any passage of air. Very little blood was lost, but just after the tumour had been removed he collapsed and died. The other (No. 363), was a female patient, aged 68, with a small substernal cystic adenoma of many years duration. She became rapidly worse during the three days that elapsed between my first seeing her and the time of the operation. Just before the operation she was in very bad condition, with a feeble and rapidly failing heart. The operation was short and easy, and attended by very little hæmorrhage. Only two drachms of chloroform were administered during the half-hour which the operation took to perform. She had been carried back to her bedroom and placed in her bed when she suddenly collapsed and died of cardiac failure, without any sign of respiratory distress. One other case (No. 135) died of another disease (carcinoma of the lung) before leaving the hospital. But as she had recovered completely from the operation I have felt justified in not placing this case among those of death from the operation. This case has already been recorded in the *Lancet*, 1902, i., p. 1227.

Malignant disease of the thyroid is but rarely seen at a period when it is suitable for operation, and the results of operation are most unsatisfactory. Only seven patients out of a large number seen were submitted to operation (Cases 162, 165, 175, 194, 233, 347 and 384). Three were advanced cases, in which operation was undertaken only in the hope of relieving urgent and severe symptoms; one of these was intrathoracic, and it was found after death that the growth had actually penetrated the trachea; all three died. In the other four the tumour was moveable, and there seemed a reasonable prospect of effecting a complete removal of the disease. In all the wounds healed quickly and well, but in three of them recurrence took place sooner or later. Case 194, a case of round-celled sarcoma, died at home three months after operation. Case 175, a spindle-celled sarcoma, was seen to be in good general health ten months after operation, but died at home five months later. Case 347, a case of carcinoma attacking a large parenchymatous goitre, was greatly relieved, and remained well for about nine months; hæmorrhage and other signs of recurrence then took place, and he is now, twelve months after the operation, again suffering severely from dyspnœa. The fourth patient, operated on only four months ago, is still well, and able to do his work as a farm labourer. These cases form but a gloomy record.

It will be seen that no cases of *genuine* exophthalmic goitre have been treated by removal.[1] The list includes, however, at least ten cases of goitre with palpitation, tremulousness, and other symptoms often found with simple goitre, as well as with Graves's disease. Such as these are by many classed as incomplete cases of Graves's disease (the so-called *formes frustes*). The advisability of the removal of the goitre of true Graves's disease is, in my opinion, still an open question. The risk of operation in true Graves's disease is undoubtedly very serious; the ultimate benefits, so far as I can learn, are by no means certain or lasting. Those who appear to have had fairly satisfactory results from the removal of goitre in cases of genuine Graves's disease are apparently very careful in the selection of the cases they submit to operation. On the other hand, operation in the cases called *formes frustes* yields excellent results, as might be expected. Detailed statistics of a long series of operations upon genuine exophthalmic goitres, giving the exact condition of each patient before, and some time after operation, are much wanted.

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[1] Three cases have been treated by ligature of thyroid arteries, but they do not come within the scope of the present paper.

Conclusions.—Operations for innocent goitre yield admirable results, and afford, as a rule, complete relief from all symptoms. The operation is, however, a delicate one, and should not be undertaken lightly, or without due attention to important details. Special attention should be paid to anæsthesia and asepsis, to the careful arrest of all hæmorrhage (especially venous), to the recurrent nerve, and to drainage for a short time. In most operations it is best not to remove that portion of the goitre that lies next to the œsophagus, recurrent nerve, and side of the trachea. The patient should be encouraged to be up and about within a very few days of the operation.

Mr. James Berry's 274 Additional Cases of Removal of Goitre by Operation.

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours.	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anæsthetic	Latest report (and source of)
127	1901 Feb. 9	Mr. C.J.E.	50	Discomfort	Enucleation left	Soft, solid adenoma, size of hen's egg	R.	No drainage, primary union	100°	1	Oblique	Chloroform	February, 1906. Quite well. (Personal observation.)
128	Mar. 19	Mrs. M.	60	Hardness, nodularity, increase in size	Extirpation right	Compound solid adenoma. Hard, irregular mass, size of hen's egg. On section, yellowish grey colour, with a good deal of fibrous tissue. Not encapsuled.	R.	Drainage 5 days, primary union	100°	2	Angular	Chloroform	Decem. 1906. Quite well. (Personal observation).
129	Apr. 15	Olive B.	27	Dyspnœa	Enucleation left	Substernal solid adenoma, size of hen's egg, extending 2 inches below upper border of sternum	R.	Drainage 1 day. Left hospital with wound apparently healed. It subsequently broke down, and patient was re-admitted with a sinus, which had to be laid open. It finally healed some months later	100·8°	2	Transverse	Chloroform	March, 1906. Quite well. (letter from Dr. Gripper, Wallington.)
130	Apr. 16	Miss D.	25	Deformity	Enucleation left	Soft, solid adenoma, size of an orange; weight 3½ ozs.	R.	Drainage, primary union	99·8°	—	Transverse	Chloroform	Novem. 1905. Quite well. (Personal observation.)
131	Apr. 20	Mary Ann P.	41	Dyspnœa	Enucleation multiple, left	Four solid adenomata; weight 16½ ozs.	R.	Drainage 1 day, primary union	101°	3	Oblique	Chloroform	February, 1906. Quite well, but remainder of goitre continues to enlarge. (Letter.)

132	May 16	Mrs. M.	39	Dyspnœa	Enucleation left	Cystic adenoma, size of hen's egg, $\frac{5}{8}$ solid; weight 2 ozs.	R.	No drainage, primary union	99·6°	—	Transverse	Chloroform	Mar. 22, 1906. Quite well. (Personal observation).
133	June 3	Lizzie W.	18	Deformity, dyspnœa	Extirpation left	Adeno-parenchymatous; weight $12\frac{1}{4}$ ozs.	R.	Drainage 1 day, primary union. Had had one previous operation at another hospital	103°	3	Oblique	Chloroform	February, 1906. Quite well. (Letter.)
134	June 4	Miss H.	39	Discomfort	Resection-enucleation right	Solid adenoma, size of hen's egg	R.	No drainage, primary union	100°	2	Transverse	Chloroform	March 7, 1906. Quite well. (Personal observation.)
135	June 17	Alice C.	47	Severe dyspnœa, cyanosis	Enucleation left	Solid adenoma of inferior horn, $1\frac{1}{2}$ inches in diameter, substernal, partly intrathoracic; the remains of a previous operation performed 7 years previously at another hospital	R.[1]	Drainage 3 days, primary union. Admitted with much dyspnœa, a left-sided goitre, extending down behind sternum, and physical signs of a mediastinal tumour. Exploratory operation, and removal of inferior horn found engaged in thorax. Wound healed normally and patient was up on the fourth day, but the dyspnœa was not relieved, and patient died 23 days after operation. Post-mortem: large secondary **sarcoma** of left lung, smaller deposits in other viscera; small primary growth in ovary.	100·2°	2	Oblique	Eucaine and morphia	
136	June 28	Victoria M.	14	Extreme dyspnœa, asphyxia, insensibility	Extirpation right and left	Parenchymatous; weight 4 ozs.	R.	Drainage 9 days, wound quite healed after 12 days.	103·6°	3	Oblique	None	February, 1907. Quite well. (Personal observation.)
137	July 8	Annie S.	53	Dyspnœa	Enucleation right	Cystic adenoma $\frac{1}{3}$ solid, size of man's fist	R.	Drainage 1 day, primary union	101°	2	Transverse	Chloroform	March, 1907. Quite well. (Personal observation.)

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[1] Recovered from operation only.

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anæsthetic	Latest report (and source of)
138	1901 July 8	Mrs. W.	55	Dyspnœa	Extirpation left	Mainly a solid adenoma, size of duck's egg, very adherent to surrounding gland; weight 4½ ozs.	R.	Drainage 1 day, wound reopened second day; secondary union. Well in three weeks	102°	4	Oblique	Chloroform	April, 1906. Quite well. (Letter.)
139	July 9	Miss A. F.	28	Discomfort, some dyspnœa, tracheal displacement	Enucleation left	Cystic adenoma, size of small orange, ⅓ solid, rest largely altered blood	R.	No drainage; small collection of blood in wound; let out 7 days after operation: wound healed a few days later. No bad symptoms at any time	100°	1	Transverse	Chloroform	June, 1906. Quite well. (Letter.)
140	July 9	Miss S.	47	Pain, recent enlargement	Enucleation left	Cystic adenoma, size of hen's egg, with recent blood extravasation	R.	No drainage, primary union	100·2°	1	Oblique	Chloroform	1907. Quite well. (Personal observation.)
141	July 29	Geo. T.	61	Rapid increase, pain, dysphagia	Enucleation right	Cystic adenoma, size of an orange; ⅓ solid	R.	Drainage 1 day, temperature then rose; drain reinserted for another 3 days; primary union	103·2°	4	Oblique	Chloroform	May 9, 1902. Quite well. (Personal observation.) March, 1906. Cannot be traced.
142	Sep. 30	Martha E.	33	Dyspnœa	Extirpation left	Parenchymatous, size of duck's egg; weight 2 ozs.	R.	Drainage 1 day, primary union	100°	1	Oblique	Chloroform	Feb., 1906. Remained quite well till June, 1905, since then a good deal of palpitation. Re-admitted. (See No. 331.)

143	Oct. 4	Edith H.	32	Dyspnœa	Enucleation right	Multilocular cystic adenoma; weight $2\frac{1}{2}$ ozs.	R.	No drainage, primary union	99·8°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
144	Oct. 7	Reg. W.	12	Dyspnœa	Resection—enucleation left	Solid adenoma of left lobe; weight 2 ozs.	R.	No drainage, primary union. The dislocation of the tumour had to be effected very rapidly as respiration ceased directly skin incision had been made	99·6°	—	Transverse	Chloroform	March, 1906. Quite well. (Dr. J. H. Pegg, Reigate.)
145	Nov. 11	Jane R.	38	Dyspnœa	Enucleation right	Solid adenoma with few small cysts, partly substernal. Size of duck's egg; weight 2 ozs.	R.	No drainage, primary union. Patient also had acromegaly	101·2	5	Oblique	Chloroform	February, 1906. Quite well. (Letter.)
146	Nov. 11	Maria M.	33	Dyspnœa	Enucleation left	Two solid adenomata size of walnut and cherry, deeply seated.	R.	No drainage, primary union	100°	1	Transverse	Chloroform	February, 1906. Cannot be traced.
147	Nov. 18	Agnes T.	37	Dyspnœa	Enucleation right	Cystic adenoma, size of hen's egg, mainly solid.	R.	No drainage, primary union	99·2°	—	Transverse	Chloroform	February, 1906. Cannot be traced.
148	Nov. 25	Annie S.	35	Dyspnœa	Resection—enucleation right	Large cystic adenoma; weight $11\frac{1}{2}$ ozs.	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	May, 1907. Quite well. (Personal observation.)
149	Dec. 9	Kate P.	23	Dyspnœa	Enucleation right	Cystic adenoma, deeply seated, $\frac{1}{2}$ fluid (recent blood), size of large walnut	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	February, 1906. Cannot be traced.
150	1902 Jan. 13	Fanny O.	36	Dyspnœa	Enucleation left	Cystic adenoma, size of duck's egg, $\frac{4}{5}$ solid	R.	Drainage 1 day, primary union	100°	2	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
151	Jan. 20	Hannah F.	45	Dyspnœa	Extirpation left	Papilliferous cystic tumour, size of large lemon	R.	Drainage 2 days, primary union. Recurrent laryngeal nerve involved in tumour and had to be cut	100·2°	2	Oblique	Chloroform	October, 1904. Quite well; voice almost normal. (Personal observation.)
152	Jan. 20	Sarah B.	42	Deformity	Enucleation left	Solid adenoma, size of walnut	R.	No drainage, primary union	99·6°	—	Transverse	Chloroform	March, 1906. Cannot be traced.

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anæsthetic	Latest report (and source of)
153	1902 Feb. 3	Fred L.	19	Dyspnœa	Extirpation right	Parenchymatous; weight 9 ozs.	R.	Drainage 1 day, primary union. Some months later a small ligature abscess and sinus, which, however, soon healed up	99·8°	—	Oblique	Chloroform	March 9, 1906. Quite well. (Personal observation.)
154	Feb. 4	Mrs. H.	44	Dyspnœa	Resection—enucleation left	Large solid adenoma, partly retroclavicular; weight 6 ozs.	R.	Drainage 3 days, primary union	100·8°	3	Transverse	Chloroform	March, 1907. Quite well. (Dr. Brash, Exeter.)
155	Feb. 17	Fanny B.	47	Dyspnœa and palpitation	Extirpation left	Solid adenoma, size small orange, deeply seated behind clavicle and sternum; weight $6\frac{1}{2}$ ozs.	R.	Drainage 2 days, primary union	99·8°	—	Oblique	Chloroform	March, 1906. Quite well. (Dr. Perram, Bedford.)
156	Mar. 17	Emily W.	43	Dyspnœa	Extirpation left, resection—enucleation right	Adeno-parenchymatous substernal, $3\frac{1}{2} \times 2\frac{1}{2}$ in.; weight $4\frac{1}{2}$ ozs.	R.	Drainage 2 days, primary union	100°	2	Vertical	Chloroform	July, 1907. Quite well. (Personal observation.)
157	Mar. 20	Miss T.	42	Dyspnœa	Enucleation right	Cystic adenoma, size of goose's egg ($\frac{1}{2}$ fluid)	R.	Drainage 1 day, primary union	99·6°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
158	Apl. 7	Gertrude I.	24	Deformity	Enucleation left	Solid adenoma, $1\frac{1}{4}$ inches in diameter	R.	Drainage 1 day, primary union	99·4°	—	Transverse	Chloroform	February, 1907 Quite well. (Letter.)
159	Apl. 14	Lizzie V.	24	Bulk, weight and slight dyspnœa	Extirpation right and middle lobes	Very large—parenchymatous; portion removed, weight 22 ozs.	D.	Drainage, accidental wound of larynx necessitating laryngotomy tube and open treatment of wound; death on eleventh day from pleurisy	103°	10	Oblique	Chloroform	—
160	Apl. 14	Madge B.	21	Dyspnœa	Resection—extirpation right	Parenchymatous; weight 8 ozs.	R.	Drainage 1 day, primary union	102°	3	Oblique	Chloroform	March, 1906. Quite well. (Letter.)

161	May 4	Mrs. R.	30	Dyspnœa	Resection—enucleation left	Solid adenoma with fibrous centre, size of goose's egg; weight 5 ozs.	R.	Drainage 2 days, primary union	100·8°	2	Transverse	Chloroform	April, 1906. Quite well. (Dr. Murdoch, Hull.)
162	May 5	Louisa H.	58	Malignancy, dyspnœa, dysphagia	Extirpation right and most of left lobes	Large bilateral malignant goitre. **Carcinoma**; weight 11 ozs.	D.	Drainage 2 days, operation very severe, followed by much collapse, and death on third day. Secondary growth in lung. For previous operation in January, 1898, see No. 53	100·8°	—	Oblique	Chloroform	—
163	May 17	Mrs. C.	50	Dyspnœa, palpitation, rapid pulse	Extirpation right	Solid adeno-parenchymatous tumour, partly substernal; weight 3½ ozs.	R.	Drainage 1 day, primary union	100°	1	Oblique	Chloroform	March, 1906. Quite well. (Personal observation.)
164	June 9	Emma D.	49	Dyspnœa	Enucleation right	Solid adenoma, size of duck's egg	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
165	June 23	Amelia H.	60	Dyspnœa, cyanosis	Enucleation (partial) right	Bilateral, largely intrathoracic **carcinomatous** tumour, extended 5½ inches below upper border of clavicle	D.	Drainage 1 day, operation very severe and incomplete; death from collapse a few hours afterwards. Post mortem showed extension of carcinomatous growth into trachea. Precise nature of growth unknown before operation	97·8°	—	Transverse	Chloroform	—
166	June 30	Florence C.	36	Dyspnœa	Enucleation right	Solid adenoma; weight 2 ozs.	R.	No drainage, primary union	99·4	—	Transverse	Chloroform	October, 1906. Quite well. (Personal observation.)
167	June 30	Annie S.	26	Dyspnœa	Enucleation right	Cystic adenoma size of small orange, half fluid	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	April, 1906. Cannot be traced.
168	July 7	Eliza P.	51	Dyspnœa	Enucleation right	Pure cyst, size of hen's egg, posterior part of gland	R.	Drainage 1 day, primary union	101°	2	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
169	July 14	Thursa B.	20	Deformity	Enucleation right	Two cystic adenomata, size of walnuts	R.	No drainage, primary union	99·6°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
170	July 14	Rose C.	43	Dyspnœa, dysphagia	Enucleation right	Cystic adenoma, size of hen's egg	R.	Drainage 1 day, primary union	100·2°	1	Transverse	Chloroform	March, 1906. Quite well.

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anæsthetic	Latest report (and source of)
171	1902 July 18	Annie N.	48	Dyspnœa	Enucleation left	Cystic adenoma, size of orange; contained 8 ozs. of fluid	R.	Drainage 2 days, primary union	101·4°	2	Transverse	Chloroform	March, 1906. Cannot be traced.
172	Sep. 15	Charles P.	57	Discomfort, increase in size	Enucleation left	Cystic adenoma, size of orange; $\frac{4}{5}$ fluid, thick fibrous wall	R.	Drainage 1 day, primary union	100°	1	Oblique	Chloroform	March 1906. Quite well. (Personal observation.)
173	Sep. 22	Mrs. S.	35	Dyspnœa	Resection—extirpation right and middle lobe	Solid adenoma; weight 7 ozs.	R.	Drainage 2 days, primary union	101·2°	2	Transverse	Chloroform	April, 1906. Quite well. (Letter.)
174	Sep. 29	Harry T.	21	Dyspnœa	Extirpation left	Parenchymatous; weight 7 ozs.	R.	Drainage 1 day, primary union	100·4°	1	Oblique	Chloroform	Decem., 1902. Quite well. (Personal observation.)
175	Sep. 29,	Joseph S.	49	Malignancy	Extirpation left	Spindle-celled **sarcoma,** size of duck's egg	R.	Drainage 1 day, primary union	98·8°	—	Oblique	Chloroform	July, 1903. In good health, but has small deep-seated recurrence. No further operation. (Personal observation.) Dec. 25, 1903. Died at home of recurrence. (Dr. Storry, Stroud.)
176	Oct. 6	Miss M. S.	21	Deformity, dyspnœa on exertion only	Enucleation (evidement) right	Cystic adenoma; weight 2 ozs.	R.	Drainage 1 day, mild suppuration and small sinus, which healed in 2 months	100°	2	Transverse	Chloroform	March, 1906. Quite well. (Dr. Baker, Rushden.)

177	Oct. 6	Emily F.	33	Dyspnœa	Enucleation right	Solid adenoma, size of walnut, at back and inner part of gland; weight ¾ oz.	R.	Drainage 1 day, primary union	100·2°	1	Transverse	Chloroform	Remained quite well as regards the neck, but died of carcinoma of the rectum in 1905. (Dr. Tolputt, Kettering.)
178	Oct. 27	Isabella C.	41	Dyspnœa	Enucleation left	Cystic adenoma, size of small orange; weight 2½ ozs.	R.	Drainage 2 days, primary union	100·8°	2	Transverse	Chloroform	February, 1906. Quite well. (Letter.)
179	Oct. 29	James G.	14	Dyspnœa	Resection—extirpation right	Parenchymatous; 2¾ × 2 inches	R.	Drainage 1 day, primary union	100°	1	Transverse with short vertical limb	Chloroform	March, 1906. Quite well. (Personal observation.)
180	Nov. 3	Avis C.	39	Dyspnœa	Enucleation right & left	4 cystic adenomata, largest size of hen's egg, ⅕ solid	R.	No drainage, primary union	99·4	—	Transverse	Chloroform	February, 1906. Quite well. (Letter.)
181	Nov. 10	Percy S.	23	Dyspnœa	Resection—extirpation right	Parenchymatous; weight 7 ozs.	R.	Drainage 1 day, primary union	99·8	—	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
182	Dec. 1	Emily E.	51	Dyspnœa	Enucleation left	Intrathoracic solid adenoma, size of small orange; weight 3 ozs.	R.	Trachea much displaced to right; drained tube and gauze for five weeks, then healed	102·4°	2	Transverse	Cocaine and Eucaine only	Sept. 1907. General health excellent but upper part of thyroid has continued to grow and recently has begun to cause dyspnœa again. Will require further operation shortly. (Personal observation.)

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anæsthetic	Latest report (and source of)
183	1902 Dec. 8	Mabel B.	17	Dyspnœa	Resection—extirpation right	Parenchymatous; 5 ozs.	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	March, 1906. Cannot be traced.
184	Dec. 8	Louisa T.	50	Dyspnœa	Enucleation right	Cystic adenoma, size of Tangerine orange, $\frac{5}{6}$ solid, with recent hæmorrhage	R.	Drainage 1 day, primary union	99·6°	—	Transverse	Chloroform	February, 1906. Quite well as regards neck, but very ill with aortic disease of heart. (Dr. Perram, Bedford.)
185	Dec. 9	Mrs. P.	55	Dyspnœa	Enucleation right	Cystic adenoma, size of small hen's egg, mainly solid, springing from back of gland.	R.	Drainage 1 day; went home with wound apparently healed by primary union; subsequently a small sinus formed which, however, soon healed	About 100° (chart lost)	1	Oblique	Chloroform	January, 1905. Quite well. (Personal observation.)
186	Dec. 12	Wm. G.	53	Dyspnœa	Extirpation right and middle	Papilliferous tumour 4 × 5 inches; weight 32 ozs.	R.	Drainage 5 days, primary union, except in track of tube. First operation	101·6°	7	Oblique	Cocaine only	See 214, 236, 280 and 326.
187	1903 Jan. 10	Miss B.	38	Discomfort, increase in size	Enucleation right	Papilliferous tumour cystic adenoma, size of hen's egg, $\frac{2}{3}$ solid	R.	No drainage, primary union	99·4°	—	Transverse	Chloroform	Sept., 1907. Quite well. (Personal observation.)
188	Jan. 12	Harriet H.	41	Dyspnœa	Resection—enucleation left	Cystic adenoma, size of lemon	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	May, 1906. Quite well. (Letter.)
189	Feb. 2	Ada P.	25	Dyspnœa	Enucleation right	Nearly pure cyst, size of hen's egg	R.	No drainage, primary union	99·2°	—	Transverse	Chloroform	March 9, 1906. Quite well. (Personal observation.)

190	Feb. 2	Ethel P.	30	Discomfort, slight dyspnœa	Enucleation right	Cystic adenoma, size of walnut; $\frac{1}{4}$ solid, with old hæmorrhage	R.	No drainage, primary union	100·2°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
191	Feb. 16	Kate L.	24	Dyspnœa	Enucleation right	Solid adenoma; weight, $1\frac{3}{4}$ ozs.	R.	No drainage, primary union	99·8°	—	Transverse	Chloroform	April, 1906. Quite well. (Dr. Stanley Smith.)
192	Feb. 23	Mary J.	24	Dyspnœa	Enucleation left	Cystic adenoma, size of hen's egg	R.	No drainage, primary union	100·6°	3	Transverse	Chloroform	Sept., 1906. Quite well. (Letter.)
193	Mar. 2	Grace M.	14	Dyspnœa	Resection right, extirpation, partial left	Parenchymatous; weight, $9\frac{1}{2}$ ozs.	R.	Drainage 7 days, mild suppuration; healed in a fortnight	101·6°	5	Transverse	Chloroform	February, 1907. Cannot be traced.
194	Mar. 3	Mrs. G.	49	Malignancy	Extirpation left	Round-celled **sarcoma**, size of hen's egg	R.	Drainage 2 or 3 days, primary union	100°	1	Oblique	Chloroform	March 16. Went home with wound healed. May 24, 1903, died at home with recurrence. (Dr. Downes, Hornsey.)
195	Mar. 23	Maud F.	38	Dyspnœa	Enucleation left	Cystic adenoma, $\frac{1}{3}$ solid; size of large walnut	R.	No drainage, primary union	100°	1	Transverse	Chloroform	March 9, 1906. Quite well. (Personal observation.)
196	Mar. 23	Walter H.	41	Hardness, increase in size, suspicion of malignancy	Resection enucleation right	Papilliferous adenoma	R.	Drainage 2 days, primary union	99·8°	—	Oblique	Chloroform	April 4. Went home quite well. March, 1906. Cannot be traced.
197	April 2	Eliza S.	53	Dyspnœa	Resection — enucleation right	Solid adenoma, $3\frac{1}{2}$ by 3 inches	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	March, 1906. General health much improved since operation, and steadily improving. (Dr. Nicholson, Hull.)

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anæsthetic	Latest report (and source of)
198	1903 May 18	Kathleen M.	28	Deformity	Enucleation right	Cystic adenoma; weight $\frac{1}{2}$ oz.	R.	No drainage, primary union	100·2°	1	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
199	May 18	Agnes W.	35	Deformity, dyspnœa on exertion	Enucleation right	Soft, solid adenoma, size of small orange; weight 4 ozs.	R.	Drainage 1 day, primary union	100·4°	2	Transverse	Chloroform	March, 1906. Cannot be traced.
200	May 18	Percy C.	17	Dyspnœa	Resection—extirpation right	Parenchymatous; weight 9 ozs.	R.	Drainage 2 days, primary union	100·4°	3	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
201	May 23	Mrs. P. F.	33	Deformity	Enucleation left	Cystic adenoma, size of walnut, left lobe	R.	Drainage 1 day, primary union	98·6°	—	Transverse	Chloroform	October, 1906. Quite well. (Personal observation.)
202	May 25	Florence D.	34	Dyspnœa	Resection—enucleation right	Two cystic adenomata, size of orange and walnut respectively	R.	Drainage several days, primary union	101°	1	Transverse	Chloroform	March 9, 1906. Quite well. (Personal observation.)
203	May 25	Ellen D.	19	Dyspnœa	Enucleation right	Soft, solid adenoma, $\frac{1}{6}$ fluid, weight $1\frac{1}{2}$ ozs.	R.	No drainage, primary union	99·6°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
204	June 4	Mrs. B.	43	Dyspnœa	Enucleation left	Solid adenoma; weight $4\frac{1}{2}$ ozs.	R.	Drainage 1 day, primary union	100·4°	2	Transverse	Chloroform	January, 1907. Quite well. (Dr. E. E. Flemming.)
205	June 15	Lilian S.	21	Deformity	Resection—extirpation left	Recurrent, papilliferous goitre; weight 7 ozs.	R.	Three previous operations at another hospital. Drainage 2 days, primary union	100·4°	2	Oblique	Chloroform	March, 1906. Quite well, but remaining gland still enlarging. (Personal observation.) For re-admission see No. 334.

206	June 15	Mary F.	20	Dyspnœa	Resection—enucleation (pyramid)	Soft, solid tumour; weight 2½ ozs.	R.	No drainage, primary union	99·8°	—	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
207	June 15	Alice B.	35	Deformity	Enucleation right	Solid adenoma at back of the gland; weight 1 oz.	R.	Drainage 2 days, primary union	100·8°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
208	June 18	Miss N.	41	Dyspnœa	Resection—enucleation right	Cystic adenoma, size of duck's egg, $\frac{1}{10}$ solid, and a solid one size of walnut	R.	No drainage, primary union	100·4°	3	Transverse	Chloroform	Decem., 1906. Quite well. (Personal observation.)
209	June 23	Miss H.	32	Dyspnœa	Resection—extirpation left	Three solid adenomata, largest size of duck's egg, partly substernal	R.	Drainage. Recurrent hæmorrhage from a superficial vein, wound partially opened up, and vein tied. Primary union except in track of drain. Wound soundly healed in 3 weeks	100·2°	1	Oblique	Chloroform	March, 1906. Quite well. (Personal observation.)
210	June 29	Elizabeth S.	42	Dyspnœa	Enucleation left	Cystic adenoma, size of small hen's egg	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	April, 1906. Cannot be traced.
211	July 6	Annie B.	28	Dyspnœa	Resection—extirpation right	Adeno-parenchymatous; weight 7 ozs.	R.	Drainage 2 days. An abscess formed after patient had left hospital; re-admitted with sinus, which closed 3½ months later	100·2°	2	Transverse	Chloroform	Nov. 20, 1903. Quite well, sinus closed. (Personal observation.) 1906. Cannot be traced.
212	July 7	Mrs. K.	36	Dyspnœa	Enucleation left	Soft solid adenoma; weight 2 ozs., with some hæmorrhage	R.	Drainage 1 day, primary union	100°	2	Transverse	Chloroform	January, 1907. Quite well. (Dr. J. L. Hewer.)
213	July 13	Mary A. S.	38	Dyspnœa	Resection—enucleation right	Large solid adenoma; weight 10½ ozs.	R.	Drainage 1 day, primary union	100·8°	3	Transverse	Chloroform	Mar. 9, 1906. Quite well. (Personal observation.)
214	July 13	Wm. G.	54	Recurrence of papilliferous tumour	Extirpation right	Recurrent papilliferous tumour; weight 8½ ozs.	R.	Drainage 1 day, primary union. Second operation	100°	1	Oblique	Chloroform	See also 186, 236, 280 and 326.

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	1903												
215	July 20	Mrs. W.	39	Dyspnœa	Enucleation right	Solid adenoma, deep-seated, size of egg	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
216	July 20	Ellen S.	45	Dyspnœa	Enucleation right and left	Small cystic adenomata, back of right and left lobes	R.	Drainage 1 day, primary union	100·2°	1	Transverse	Chloroform	April, 1906. Cannot be traced.
217	Aug. 31	Maud B.	24	Dyspnœa	Enucleation right	Cystic adenoma, size of Tangerine orange	R.	No drainage, primary union	99·4°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
218	Sept. 7	Kate T.	34	Increase in size	Enucleation right	Solid adenoma, size of Tangerine orange; weight 2 ozs.	R.	Drainage 2 days, primary union	99·6°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
219	Sept. 7	Kate C.	24	Dyspnœa	Enucleation right	Cystic adenoma with old hæmorrhage; weight 2 ozs.	R.	Drainage 1 day, primary union	99·2°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
220	Sept. 14	Emma S.	39	Dyspnœa	Enucleation (evidement) right	Large soft solid adenoma	R.	Drainage 1 day, primary union	100·2°	1	Transverse	Chloroform	1905. Quite well. (Personal observation.)
221	Sept. 17	Mrs. S.	30	Deformity	Resection—enucleation right and small part of left	Adeno-parenchymatous goitre, mainly right lobe; weight 11 ozs.	R.	Drainage 1 day, primary union	100·4°	1	Transverse	Chloroform	February, 1906. Quite well. (Letter.)
222	Oct. 12	Edith W.	27	Increase in size	Enucleation left	Cystic adenoma, size of Tangerine orange; weight 2½ ozs.	R.	Drainage 1 day, primary union	100·2°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
223	Oct. 12	Eliza S.	36	Dyspnœa, emaciation, rapid pulse (120—160)	Resection—enucleation right	Fœtal adenoma size of duck's egg	R.	Drainage 5 days, primary union except in track of drain, healed in a few days	102·8°[1]	3	Transverse	Chloroform	March, 1906. Quite well. (Letter.)

[1] Temperature 100° before operation.

224	Oct. 19	Lizzie R.	38	Dyspnœa	Resection—extirpation left, enucleation adenoma of pyramid	Mainly parenchymatous, but with solid adenoma in left lobe; weight 4½ ozs.	R.	Drainage 1 day, primary union	99·6°	—	Transverse	Chloroform	March, 1906. Quite well, but right lobe now enlarged to size of goose's egg. (Personal observation.)
225	Oct. 20	Miss S.	37	Deformity, dyspnœa on exertion	Enucleation left	Solid adenoma, size of goose's egg; weight 5 ozs.	R.	Drainage 1 day, apparent primary union, then late subcutaneous suppuration, small sinus for 4 weeks	100·4°	1	Transverse	Chloroform	1907. Quite well. (Personal observation.)
226	Oct. 26	Ethel N.	26	Dyspnœa	Enucleation left	Solid adenoma; weight 3 ozs.	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
227	Oct. 30	Janet T.	27	Increase in size	Enucleation right	Solid adenoma, size of pigeon's egg	R.	No drainage, primary union	100°	1	Transverse	Chloroform	March, 1906. Cannot be traced.
228	Nov. 10	Miss V.	36	Dyspnœa	Resection—enucleation left	Cystic adenoma, size of orange, ⅓ solid; weight 6 ozs.	R.	Drainage 2 days, primary union	99·8°	—	Transverse	Chloroform	January, 1907. Quite well. (Letter.)
229	Nov. 10	Miss V.	39	Deformity	Enucleation left	Solid adenoma, size of large walnut, left lobe; weight 1 oz.	R.	No drainage, primary union	100°	1	Transverse	Chloroform	January, 1907. Quite well. (Letter.)
230	Nov. 11	Mrs. C.	23	Deformity	Enucleation left	Cystic adenoma, size of an egg, ¾ fluid, mainly altered blood	R.	Drainage 1 day, primary union	99·2°	—	Transverse	Chloroform	March 1906. Quite well. (Dr. M. Prickett.)
231	Nov. 23	Mabel McL.	29	Dyspnœa	Resection—enucleation right	Multiple cystic adenomata. Main tumour size of orange, very deep, extending well into thorax, very adherent to surrounding tissues	R.	Drainage 14 days, primary union, except in track of tube, where sinus persisted for another 10 days	101°	3	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
232	Dec. 8	Miss W.	38	Dyspnœa	Resection—extirpation right Resection—enucleation left	Adeno-parenchymatous. Right lobe, 3½ ozs.; left lobe, 2 ozs.	R.	Drainage about 2 days, primary union, part of right lobe removed from within the thorax	100°	1	Transverse	Chloroform	April, 1906. Quite well. (Dr. John Scott, Bromley.)

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anaesthetic	Latest report (and source of)
	1904												
233	Jan. 11	Sarah B.	43	Dyspnœa, malignancy	Extirpation right	**Carcinoma**, $4\frac{1}{2} \times 5\frac{1}{2}$ in., weight 25 ozs. Hard, irregular tumour, size of fœtal head; 15 years duration, recently become malignant	D.	Operation very severe; undertaken only at express wish of patient; tumour very large and fixed, sternum infiltrated with new growth. Death from shock 50 mins. after operation	—	—	Transverse	Chloroform	—
234	Jan. 26	Mary S.	27	Deformity, slight dyspnœa	Enucleation left	Cystic adenoma, size of hen's egg	R.	No drainage, primary union	99·4°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
235	Feb. 4	Miss D.	38	Deformity	Extirpation (pyramid only)	Cystic adenoma, size of walnut, in middle line, just over larynx	R.	No drainage, primary union	98·6°	—	Transverse	Chloroform	October, 1906. Quite well. (Personal observation.)
236	Feb. 11	Wm. G.	54	Recurrence of papilliferous tumour	Extirpation most of left lobe	Large recurrent papilliferous tumour	R.	Drainage for a day or two. Wound said to have broken down after leaving hospital, but soon healed. Third operation	102	4	Oblique	Chloroform	(See 186, 236, 280 and 326.)
237	Feb. 15	Geoffrey R.	17	Dyspnœa	Resection—extirpation bilateral, right and left	Parenchymatous goitre; weight $8\frac{1}{2}$ ozs.	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	May, 1906. Quite well. (Personal observation.)
238	Feb. 22	Miss E. M. R.	38	Dyspnœa	Enucleation right	Cystic adenoma, size of orange, with much extravasated blood	R.	Drainage 1 day, primary union	100·2°	1	Transverse	Chloroform	October, 1905. Quite well. (Sister.)
239	Feb. 22	Emma P.	44	Dyspnœa	Enucleation right	Cystic adenoma, size of hen's egg.	R.	No drainage, primary union	99·6°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
240	Feb. 29	Lucy D.	31	Dyspnœa	Enucleation left	Cystic adenoma (inflamed), 2 in. in diameter	R.	Drainage 2 days, primary union	99·2°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)

241	Mar. 14	Ellen R.	15	Deformity, slight dyspnœa	Enucleation right	Soft solid adenoma, size of walnut	R.	No drainage, primary union	99°		Transverse	Chloroform	1906. Quite well. (Dr. Square, Leighton Buzzard.)
242	Mar. 28	Florence W.	22	Discomfort, slight dyspnœa	Enucleation right	Cystic adenoma; weight 1½ ozs.	R.	No drainage, primary union	100°	2	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
243	April 7	Miss M.A.L.	60	Dyspnœa	Enucleation left	Solid adenoma, size of an orange, almost wholly intrathoracic, left lobe. Above this a smaller one, size of walnut; weight 3¾ ozs.	R.	Drainage 4 weeks (tube and gauze 2 weeks, gauze only 2 weeks)	100·6°	1	Transverse	Chloroform	July, 1907. Quite well, but still has slight stridor, the long compressed trachea having not yet quite regained its normal shape. (Personal observation.)
244	April 7	Mr. J.S.	63	Dyspnœa	Enucleation left	Two adenomata right lobe, size of small oranges, one deeply seated, partly substernal	R.	Drainage for 4 days, primary union except in track of drain. Had a little bronchitis for a few days after operation	102·4°	5	Transverse	Chloroform	June, 1905. Quite well. (Personal observation.) Jan. 21, 1907. Died of apoplexy after a two days' illness; had remained quite well till then. (Dr. P. MacGregor, Huddersfield.)
245	April 25	Sarah S.	22	Deformity, slight increasing dyspnœa	Enucleation right	Cystic adenoma, size of small orange, containing mainly viscid yellow colloid	R.	Went home after operation, apparently quite well; readmitted a few days later with a small painless abscess under the scar; opened, healed in a few days	99·4°	—	Transverse	Chloroform	January, 1906. Quite well. (Personal observation.)

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246	1904 April 26	Mrs. C.	45	Dyspnœa, deformity	Resection—enucleation left	Solid adenoma, weighing 11 ozs.	R.	Drainage 4 days, primary union	100·2°	1	Transverse	Chloroform	October, 1906. Quite well. (Personal observation.)
247	May 2	Agnes A.	49	Dyspnœa	Resection—enucleation right	Two solid adenomata size of oranges, one deeply seated, partly substernal; weight 14 ozs.	R.	Drainage 2 days, primary union	99·4°	—	Transverse	Chloroform	February, 1907. Quite well. (Dr. L. A. Winter, Farnborough.)
248	May 3	Mr. T.P.W.	37	Dyspnœa	Enucleation right and left	Both main tumours, wholly intrathoracic (below level of first rib); right, solid and partly calcified adenoma, size of large orange; left, rather smaller, mainly solid, but with about 2 ozs. of clear fluid as well. Two other solid tumours as large as walnuts also removed from right and left lobes respectively	R.	A long and difficult operation, as the veins about the root of the neck were very numerous and much distended from long-continued pressure. Most of the solid contents of the right tumour had to be scooped out and all the fluid contents of the left tumour evacuated before either tumours could be drawn up through the upper opening of the thorax and enucleated. After operation wound drained for 3 days with tube and gauze, then two days with gauze alone. Pulse never above 78. Patient up on 3rd day, out of doors on 6th day; never had a bad symptom in spite of the temperature; went home to his own doctor on the 9th day, with superficial granulating wound	102°	4	Transverse	Chloroform	February, 1907. Quite well. (Dr. Barton, Wimbledon.)

249	May 9	Carrie S.	25	Deformity slight but increasing dyspnœa	Enucleation right	Two solid adenomata, one size of walnut at apex of upper horn, the other size of duck's egg at inferior horn, both of right lobe	R.	Drainage 1 day, primary union	101·6°	2	Transverse. One incision only for both tumours	Chloroform	March, 1906. Cannot be traced.
250	May 10	Emily H.	35	Deformity	Enucleation left	Soft solid adenoma; weight 1 oz.	R.	No drainage, primary union (second operation). For previous operation see No. 109	99°	—	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
251	June 14	Mrs. J.	44	Pain, discomfort, possible malignancy	Enucleation right	Old fibroid cystic adenoma, back of right lobe, size of walnut, recent accumulation of fluid.	R.	Drainage 1 day, primary union	99	—	Transverse (in posterior triangle)	Chloroform	August, 1906. Quite well. (Dr. H. Troutbeck, Westminster.)
252	June 21	Miss Edith J.	30	Dyspnœa	Resection—enucleation left	Cystic adenoma, mainly solid, size of large orange, partly substernal.	R.	Drainage 3 days, primary union	99°	—	Transverse	Chloroform	June, 1905. Quite well. (Personal observation.)
253	June 22	Mrs. M.	57	Nocturnal dyspnœa, possible malignancy	Resection—enucleation right	Old cystic adenoma, size of walnut, at back and inner part of right lobe.	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	1907. Quite well. (Personal observation.)
254	June 27	Philip P.	56	Dyspnœa (20 years or more)	Resection—enucleation right	Main tumour solid adenoma, size of goose's egg, $\frac{2}{3}$ substernal, $\frac{1}{2}$ intrathoracic, also 2 other solid adenomata, size of walnut, enucleated from upper part of same lobe; weight 3 ozs.	R.	Drainage 22 days; tumour easily pulled up out of thorax, very little hæmorrhage. Went home 31st day quite well.	101·8°	2	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anæsthetic	Latest report (and source of)
255	1904 July 4	Dor'thea P.	23	Deformity, slight dyspnœa	Enucleation right	Three small cystic adenomata, with hæmorrhage into one	R.	Drainage 1 day, primary union. Patient had mitral disease and rapid pulse before operation	100°	1	Transverse	Chloroform	March, 1907. Quite well. (Dr. Barber, Hastings.)
256	July 8	John B.	21	Dyspnœa	Enucleation left	Cystic adenoma, size of hen's egg, left lobe and isthmus; weight $3\frac{1}{2}$ ozs.	R.	Drainage 1 day, primary union	99·6	—	Transverse	Chloroform	April, 1906. Cannot be traced.
257	July 9	Miss G.	28	Deformity, slight dyspnœa	Enucleation right	Soft solid adenoma, size of bantam's egg	R.	Drainage 1 day, primary union	100·2° (T 100° P 120 before operation)	1	Transverse	Chloroform	February, 1907. Quite well. (Letter.)
258	July 12	Edith N.	32	Dyspnœa	Resection—extirpation left	Parenchymatous with multiple adenomata; weight $7\frac{1}{2}$ ozs.	R.	Drainage 1 day; left hospital; wound apparently healed by primary union; abscess subsequently, and sinus for several weeks	100·8°	2	Transverse	Chloroform	March, 1906. Quite well. (Dr. Tweedie, Market Harborough
259	July 12	Mrs. H.	31	Deformity, slight dyspnœa	Enucleation right	Soft solid adenoma, size of hen's egg	R.	Drainage 12 hours, primary union	99°	—	Transverse	Chloroform	1905. Quite well. (Personal observation.)
260	July 18	Loveday W.	33	Dyspnœa	Resection—extirpation right	Parenchymatous goitre, with 2 small solid adenomata; weight $2\frac{1}{2}$ ozs.	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	April, 1906. Quite well. (Letter.)
261	July 18	Emma P.	43	Dyspnœa	Resection—enucleation right	Multiple cystic adenomata; weight 6 ozs.	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	July, 1906. Quite well. (Dr. Dryland, Kettering.)

262	Aug. 2	Esther C.	42	Dyspnœa	Resection—extirpation right	Multiple solid adenomata; weight 6 ozs.	R.	Drainage 6 days, primary union	101°	3	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
263	Oct. 4	Annie L.	20	Deformity, dyspnœa	Enucleation left	Cystic adenoma, with hæmorrhage, size of orange	R.	Drainage 1 day, primary union	99·4°	—	Transverse	Chloroform	May, 1907. Quite well. (Personal observation.)
264	Oct. 7	Annie B.	32	Dyspnœa	Enucleation left	Cystic adenoma, size of orange	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	April, 1906. Quite well. (Letter.)
265	Oct. 10	Ellen B.	28	Deformity	Enucleation right	Solid adenoma, size of hen's egg	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
266	Oct. 10	Charlotte F.	23	Deformity, dyspnœa	Extirpation right	Parenchymatous goitre; weight 7 ozs.	R.	Drainage 1 day; on 7th day temp. rose to 100°, on 8th day to 101°; wound was then opened up and some thin purulent fluid was let out. Patient never very ill; vocal cord not moving freely	101°	4	Transverse	Chloroform	February, 1907. Quite well. (Dr. E. W. Cross, Leytonstone.)
267	Oct. 14	Mary B.	38	Deformity, slight dyspnœa	Resection—enucleation left	Soft solid adenoma; weight 7 ozs.	R.	Drainage 1 day, primary union	99·6°	—	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
268	Oct. 24	Albert C.	21	Deformity	Enucleation multiple right	Cystic adenomata, size of large walnut; 2 others size of cherries	R.	Drainage 2 days, primary union	99·8°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
269	Oct. 11	Edith P.	36	Deformity	Resection—enucleation right	Cystic adenoma, weight 5½ ozs.	R.	Drainage 1 day, primary union	100·4°	1	Transverse	Chloroform	Novem., 1904. Quite well (Personal observation.) 1906. Cannot be traced.
270	Oct. 31	Ellen R.	21	Dyspnœa	Extirpation right	Parenchymatous goitre; weight 7 ozs.	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)

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271	1904 Nov. 7	Mabel B.	33	Discomfort and dyspnœa	Resection—enucleation right	Multilocular cystic adenoma, size of an egg, partly substernal, springing from back of, and lower part of, right lobe	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	Remained quite well and free from all trouble till June, 1905; then had hæmoptysis and pneumonia, and died July 1905; she had had symptoms of phthisis for some years.
272	Nov. 14	Annie B.	44	Dyspnœa, deformity	Resection—enucleation left	Three solid adenomata; weight 19½ ozs.	R.	Drainage many days. Post-operative effusion of blood into wound. Late suppuration, sinus lasting several weeks	102·4°	Several weeks	Transverse	Chloroform	May, 1905. Quite well. (Dr. W. L. Stuart, Camberley.)
273	Nov. 14	Eliza P.	50	Dyspnœa, sudden increase in size	Enucleation right	Cystic adenoma, size of large walnut	R.	No drainage, primary union	100°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
274	Nov. 21	Daisy W.	27	Deformity	Enucleation (atypical) right	Parenchymatous, with adenomata; weight 2 ozs.	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	January, 1907. Quite well. (Letter.)
275	Nov. 22	Mrs. D.	49	Deformity, discomfort	Enucleation right	Solid adenoma; weight 5½ ozs.	R.	Drainage 1 day, primary union, temperature for a week just over 100° but no local or general symptoms to account for this. Pulse 80—90 throughout	101°	7	Transverse	Chloroform	Dec., 1905. Quite well. (Personal observation.)

276	Nov. 28	Annie J.	46	Dyspnœa	Resection—extirpation right and part of left	Parenchymatous, with adenomata and fibrosis; weight 9 ozs.	R.	Drainage 1 day, primary union (small stitch abscess at one point)	100·8°	7	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
277	Dec. 5	Mary M.	24	Deformity	Enucleation right	Cystic adenoma, $2\frac{1}{4} \times 1\frac{1}{4}$ inches	R.	No drainage, primary union	100·2°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
278	Dec. 5	Eliz'be'h S.	28	Deformity	Resection enucleation right	Solid adenoma, $2\frac{3}{4} \times 2\frac{1}{2}$ inches; weight 4 ozs.	R.	Gauze drain 14 days, healing delayed by hæmorrhage into wound, discharged 18 days after operation, wound healed, no suppuration	100·2°	2	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
279	Dec. 12	Maud T.	23	Deformity	Enucleation (evidement) right	Soft solid adenoma; weight 2 ozs.	R.	Drainage 1 day, primary union	100°	3	Transverse	Chloroform	June 27, 1905. Quite well. (Personal observation.)
280	Dec. 12	Wm. G.	57	Recurrence of papilliferous tumour	Extirpation right	Three recurrent papilliferous tumours; weight 4 ozs.	R.	Drainage 2 days, primary union (fourth operation)	101·4°	1	Oblique	Chloroform	*See* Nos. 186, 214, 236, and 326.
281	1905 Jan. 9	Emma S.	32	Deformity	Enucleation right	Solid adenoma; weight 3 ozs.	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	March, 1906. Quite well (Letter.)
282	Jan. 10	Mrs. B.	25	Discomfort, dyspnœa, dysphagia	Enucleation pyramid and left	Two small solid adenomata, one at inner and back part of left lobe	R.	Drainage 1 day, primary union	99·6°	—	Transverse	Chloroform	Feb. 14, 1906. Quite well. (Personal observation.)
283	Jan. 16	Rudolf M.	25	Rapid growth, suspected malignancy	Resection—extirpation left	Suppurating **hydatid cyst**	R.	Drainage 1 day, primary union	99·8°	—	V shap'd	Chloroform	March, 1906. Cannot be traced.
284	Jan. 30	Mary P.	50	Dyspnœa	Enucleation left	Solid adenoma, $3\frac{1}{2} \times 2\frac{1}{2}$ inches; weight 6 ozs., fibrous in centre	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	Feb. 2, 1906. Quite well. (Personal observation.)
285	Jan. 30	Ethel H.	50	Deformity	Enucleation right	Solid adenoma; weight 1 oz.	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)

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286	1905 Feb. 2	Mr. C.G.C.	47	Dyspnœa	Resection—enucleation left	Solid adenoma, size of emu's egg; weight 9 ozs.	R.	Drainage 2 days, primary union	100°	1	Transverse	Chloroform	January, 1907. Quite well. (Letter.)
287	Feb. 13	Fanny W.	30	Dyspnœa	Extirpation right, enucleation left	Parenchymatous goitre, with adenomata; weight, 8 ozs.	R.	Drainage 1 day, primary union	99·4°	—	Transverse	Chloroform	March, 1906. Quite well. (Letter.)
288	Feb. 15	Sidney M.	17	Dyspnœa	Resection—extirpation right & left	Parenchymatous; weight 10 ozs.	D.	Patient pale and thin, in extremely bad condition before operation, from severe dyspnœa and orthopnea. Operation without general anæsthesia, with patient sitting upon table. Tracheal walls almost in contact. Sudden collapse just after removal of the goitre. Very little blood lost during operation.	100.6°	2	Transverse	Eucaine only	
289	Feb. 16	Miss H.	18	Deformity	Resection—enucleation right	Congenital cystic adenoma, partly hæmorrhagic, size of orange	R.	Drainage 1 day, primary union	100·6°	2	Transverse	Chloroform	Nov., 1906. Quite well. Both lateral lobes have continued to grow, but do not cause any trouble as yet. (Personal observation.)
290	Feb. 21	Mrs. W.	55	Discomfort, deformity, recent increase in size	Resection—enucleation left	Solid adenoma, left upper horn, size of orange	R.	Drainage 2 days, primary union	100°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)

291	Mar. 6	Ivy C.	16	Dyspnœa	Enucleation (atypical) right	Parenchymatous with adenomata; weight 2 ozs.	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
292	Mar. 7	Miss Daisy T.	23	Deformity, slight dyspnœa	Enucleation right	Solid adenoma, size of small orange	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	June, 1907. Quite well. (Personal observation.)
293	Mar. 20	Mary P.	56	Dyspnœa	Resection—enucleation right	Two cystic adenomata $\frac{1}{3}$ solid, size of orange and of a large walnut	R.	Drainage 1 day, primary union	100·6°	4	Transverse	Chloroform	February, 1907. Quite well. (Dr. Maude, Westerham.)
294	Mar. 21	Amy B.	22	Severe and sudden dyspnœa	Resection—enucleation left	Soft, solid, substernal adenoma with recent hæmorrhage, size of duck's egg	R.	Drainage 1 day, primary union	100·4°	1	Transverse	Chloroform	March, 1907. Quite well. (Letter.)
295	Mar. 27	Miss Helena R.	33	Dyspnœa, palpitation	Resection—enucleation right	Cystic adenoma, size of orange, $\frac{4}{5}$ fluid	R.	Drainage 1 day, primary union. Patient had a dilated heart	100·4°	3	Transverse	Chloroform	Oct. 23, 1905. Quite well. Heart symptoms entirely gone. (Personal observation.)
296	April 3	Mrs. S.	39	Discomfort, increase in size	Resection—enucleation left, enucleation right	Three cystic adenomata mainly solid, size of large walnut (left) and 2 cherries (right)	R.	Drainage 1 day, primary union	100·4°	2	Transverse	Chloroform	February, 1907. Quite well. (Personal observation.)
297	April 3	Allan H.	28	Dyspnœa	Resection—enucleation left	Cystic adenoma, size of emu's egg (nearly pure cyst)	R.	Some blood collected in wound, and required gauze drain for several days; primary union	99·8°	—	Transverse	Chloroform	February, 1907. Quite well. (Letter.)
298	May 2	Mrs. C.	46	Dyspnœa and cardiac symptoms, palpitation, &c.	Resection—enucleation right, enucleation left and pyramid	Three solid adenomata, largest size of an orange, in right inferior horn	R.	Drainage 2 days, primary union; extremely irregular pulse before operation	99°		Transverse with short vertical	Chloroform	May, 1905. Quite well; pulse regular and of good volume. May, 1906. Quite well. (Letter.) 1907. Gone back to Australia.

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299	1905 May 29	Matilda P.	30	Dyspnœa	Enucleation left	Solid adenoma, weight $1\frac{1}{2}$ ozs.	R.	Drainage 1 day, primary union	100°[1]	1	Trans verse	Chloroform	February, 1907. Quite well. (Letter.)
300	June 5	Mr. Wm. C.	36	Dyspnœa	Resection—enucleation right	Solid adenoma, size of goose's egg; weight $6\frac{1}{4}$ ozs.	R.	Drainage 2 days, primary union	99·8°	—	Transverse	Chloroform	February, 1907. Quite well. (Dr. Drummond-Morier.)
301	June 5	Emily G.	38	Dyspnœa	Resection—extirpation right	Adenoparenchymatous; weight 1 oz.	R.	Drainage 1 day, primary union	99·8°	—	Transverse with short vertical limb	Chloroform	Nov., 1906. Quite well. (Personal observation.)
302	July 14	Walter W.	24	Discomfort	Enucleation left	Solid adenoma, size of walnut	R.	Drainage 1 day, primary union	100°	1	Transverse	Chloroform	Sept. 20, 1907. Quite well. (Letter.)
303	July 17	Jane M.	30	Dyspnœa	Resection—enucleation right	Multiple cystic adenomata, size of walnut	R.	Drainage 1 day, primary union	99·6°	—	Transverse	Chloroform	February, 1907. Quite well. (Personal observation.)
304	Sept. 22	Hannah R.	56	Slight dyspnœa	Resection—enucleation right	Solid adenoma; weight 6 ozs.	R.	Drainage 1 day; readmitted in October with mild suppuration; wound soundly healed by November 8	100·4°	3	Transverse	Chloroform	February, 1907. Quite well. (Personal observation.)
305	Sept. 29	Ada S.	25	Deformity	Resection—enucleation (pyramid)	Cystic adenoma, size of pigeon's egg	R.	No drainage, primary union	99·8°	—	Transverse	Chloroform	March, 1906. Quite well. (Personal observation.)
306	Oct. 2	Eliza M.	31	Deformity	Enucleation left and right	Three solid tumours, partly calcified; weight 3 ozs.	R.	Drainage 2 days, primary union	101°	4	Transverse	Chloroform	April, 1906. Quite well. (Letter.)

[1] 100° before operation.

307	Oct. 9	Agnes H.	30	Dyspnœa	Resection—extirpation right	Parenchymatous, weight $2\frac{1}{2}$ ozs.	R.	Drainage 1 day, apparent primary union; readmitted a few days later with a superficial abscess, which soon healed	100·2°	1	Transverse	Chloroform	May 24, 1907. Quite well as regards thyroid and scar but general health not good. She has phthisis and tuberculous glands of the neck. (Personal observation.)
308	Oct. 9	Wm. S.	15	Dyspnœa	Resection—enucleation left	Cystic adenoma, retro-clavicular and retro-sternal; weight 3 ozs.	R	Drainage 2 days, primary union	100·2°	1	Transverse	Chloroform	May, 1906. Quite well. (Dr. Hoole, Parwich.)
309	Oct. 16	Clara W.	30	Dyspnœa	Resection—enucleation right	Partly calcified solid adenoma; weight 3 ozs.	R.	Drainage 1 day, primary union	100·6°	2	Transverse	Chloroform	February, 1906. Quite well. (Personal observation.)
310	Oct. 16	Ada D.	36	Dyspnœa	Resection extirpation right and part of left	Parenchymatous, with fibrosis; weight $9\frac{1}{2}$ ozs.	R.	Drainage 2 days, primary union; has also mitral disease	100·2°	1	Transverse	Chloroform	February, 1907. Quite well. (Letter.)
311	Oct. 23	Amy A.	40	Dyspnœa	Enucleation right and part of left	Three very soft solid adenomata, size of walnut and cherries	R.	Drainage 2 days, primary union	99·8°	1	Transverse	Chloroform	Sept., 1907. Quite well. (Personal observation.)
312	Nov. 6	Bessie P.	32	Dyspnœa, rapid pulse	Resection—extirpation right	Adeno-parenchymatous, with intra-thoracic inferior horn, $4\frac{1}{2} \times 3 \times 2$ ins.; weight 7 ozs.	R.	Drainage 2 days, primary union	100°	1	Transverse	Chloroform	February, 1907. Quite well. (Personal observation.)
313	Nov. 6	Kate E.	37	Dyspnœa	Enucleation right and left	Two old cystic adenomata, size of walnut and small orange, deeply seated	R.	Drainage 2 days, primary union	100·2°	2	Transverse	Chloroform	February, 1907. Quite well. Has returned to India. (Letter.)

No.	Date of operation	Name	Age	Chief reason for operation	Operation	Nature of goitre	Result	Remarks	Highest temp. after operation. Taken every 4 hours	Number of days after operation on which temp. reached or exceeded 100°	Incision	Anæsthetic	Latest report (and source of)
314	1905 Nov. 29	Mrs. B.	42	Dyspnœa	Enucleation right	Solid adenoma at back of gland, partly substernal; weight 3 ozs.	R.	Drainage about 2 days, primary union	100·2°	3	Transverse	Chloroform	Dec., 1906. Quite well. (Personal observation.)
315	Nov. 30	Mrs. H.	47	Dyspnœa	Resection—enucleation left	Adenoma, mainly cystic, size of goose's egg	R.	Drainage 2 days, primary union	99·8°	—	Transverse	Chloroform	February, 1907. Quite well. (Dr. Vernon, Ashford.)
316	Dec. 4	Albina T.	14	Dyspnœa	Resection—enucleation right	Adenoma, parenchymatous, size of a hen's egg	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	February, 1906. Quite well. (Letter.)
317	1906 Jan. 23	Miss Louie J.	43	Dyspnœa	Resection—enucleation right	Solid adenoma, with fibrous centre; weight 6 ozs.	R.	Drainage 2 days, primary union	100·6°	1	Transverse	Chloroform	February, 1907. Quite well. (Personal observation.)
318	Jan. 29	Emily B.	15	Dyspnœa	Resection—right	Parenchymatous goitre, $3\frac{1}{2} \times 2\frac{1}{2} \times 1\frac{1}{2}$ inches; weight 4 ozs.	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	February, 1907. Quite well. (Personal observation.)
319	Feb. 5	Prudence F.	36	Dyspnœa	Resection—right and left, enucleation left	Four cystic adenomata, largest size of duck's egg, one substernal	R.	Drainage 2 days, primary union	100°	1	Transverse	Chloroform	Feb. 8, 1907. Quite well. (Personal observation.)
320	Feb. 12	Amy G.	39	Discomfort, slight dyspnœa	Resection—enucleation right	Solid adenoma, size of large walnut	R.	No drainage, primary union	99·4°	—	Transverse	Chloroform	February, 1907. Quite well. (Letter.)
321	Feb. 17	Mabel B.	21	Dyspnœa	Enucleation (atypical) right	Adeno-parenchymatous goitre, size of walnut	R.	Drainage 2 days, primary union	99·2°	—	Transverse	Chloroform	February, 1907. Quite well. (Letter.)

322	Feb. 17	May O.	33	Dyspnœa	Resection—enucleation left	Compound solid adenoma; weight 4 ozs.	R.	Drainage 2 days, primary union	98·8°	—	Transverse	Chloroform	Sept., 1907. Quite well. (Personal observation.)
323	Feb. 26	Lucie B.	33	Dyspnœa	Resection—enucleation left	Adeno-parenchymatous, mainly substernal; weight 5 ozs.	R.	Drainage 4 days, primary union	100°	1	Transverse with short vertical limb	Chloro form	June, 1907. Quite well. (Personal observation.)
324	Feb. 26	Emily S.	47	Dyspnœa	Multiple resection—enucleation right and left	Adeno-parenchymatous; weight 4½ ozs.	R.	Drainage 2 days, primary union	100°	2	Transverse	Chloroform	February, 1907. Quite well, but with a small sinus. (Personal observation.) Sept., 1907. Quite well, sinus quite healed. (Personal observation.)
325	Mar. 5	Emily A.	35	Dyspnœa	Resection—enucleation right	Solid soft adenoma, size of large orange; weight 7½ ozs.	R.	Drainage 2 days, primary union	100°	2	Transverse	Chloroform	February, 1907. Quite well (Dr. Milestone, Sheffield.)
326	Mar. 5	Wm. G.	58	Recurrence papilliferous tumour	Extirpation left	Solid papilliferous tumour, size of duck's egg	R.	Drainage 1 day, primary union. Fifth operation	99·8°	—	Oblique	Chloroform	October, 1906. General health fairly good, but has further recurrence (Dr. J. Farrar, Gainsboro'.)
327	Mar. 5	Betsy H.	48	Huge size, dyspnœa	Extirpation left and part of right	Large solid adenoma; weight 12 ozs.	R.	Drainage 10 days, primary union, except in track of tube, where a small sinus persisted for about two months	101·4°	8	Oblique	Chloroform	October, 1906. Quite well. (Dr. Withers, Horncastle.)
328	Mar. 19	Ellen G.	43	Dyspnœa	Resection—enucleation left and middle	Two solid adenomata, size of hen's eggs	R.	Drainage 2 days, primary union	100°	1	Transverse	Chloroform	February, 1907. Quite well. (Dr. Vernon, Ashford.)

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329	1906 Mar. 26	May C.	30	Dyspnœa	Resection—enucleation right	Solid adenoma, size of hen's egg	R.	Drainage 2 days, primary union	99·4°	—	Transverse	Chloroform	February, 1907. Quite well. (Letter.)
330	April 2	Jane R.	42	Bulk, weight and deformity, with slight dyspnœa	Resection—extirpation right and part of left	Parenchymatous, with commencing cystic adenomata; weight $9\frac{1}{2}$ ozs.	R.	Drainage 2 days, primary union	99·2°	—	Transverse	Chloroform	Nov., 1906. Quite well. (Personal observation.)
331	April 2	Martha E.	37	Dyspnœa, palpitation	Resection—extirpation right	Parenchymatous; weight $\frac{1}{2}$ oz.	R.	Drainage 1 day, primary union. Second operation. (*See* No. 142)	100·2°	2	Transverse	Chloroform	Sept., 1907. Quite well. (Dr. A. P. Steavenson.)
332	April 9	Marg'r't L.	25	Deformity, slight dyspnœa	Resection—extirpation right	Parenchymatous; weight $9\frac{1}{2}$ ozs.	R.	Drainage 2 days, primary union. Second operation. (*See* No. 36)	100·4°	1	Transverse	Chloroform	May, 1907. Quite well. (Personal observation.)
333	May 27	Wm. S.	19	Dyspnœa	Resection—extirpation right	Parenchymatous, $5\frac{1}{2} \times 2 \times 2$ inches; weight, $5\frac{1}{2}$ ozs.	R.	Drainage 1 day, primary union, except in track of drain. All soundly healed by twenty-third day. Some months later an abscess formed, which was opened, and soon healed up	100°	1	Transverse	Chloroform	February, 1907. Quite well. (Letter.)
334	May 27	Lillian S.	24	Recurrence	Enucleation multiple right	Five papilliferous cystic adenomata; weight $1\frac{3}{4}$ ozs.	R.	Drainage 2 days, primary union. (See No. 205)	99·4°	—	Transverse	Chloroform	February, 1907. Quite well, but growth recurring again; now size of duck's egg. (Personal observation.) (Dr. Harold.)

335	May 29	Miss G. M.	28	Dyspnœa	Resection—enucleation right	Cystic adenoma, size of hen's egg; weight $2\frac{1}{2}$ ozs., with some calcification	R.	Drainage 2 days, primary union	99·4°	—	Transverse	Chloroform	October, 1906. Quite well. (Personal observation.)
336	June 9	Mrs. A.	46	Dyspnœa	Resection—enucleation right	Two solid adenomata; weight $8\frac{1}{2}$ ozs.	R.	Drainage 1 day, primary union	100·2°	1	Transverse	Chloroform	Sept. 17, 1907. Quite well. (Dr. J. R. Kingdon, King's Lynn.)
337	June 11	Mary Ann S.	38	Dyspnœa	Enucleation right & left (multiple)	Six soft solid adenomata, 5 from right lobe, 1 from left	R.	Drainage 2 days, primary union	98·8°	—	Transverse	Chloroform	July, 1907. Quite well. (Letter.)
338	June 17	Miss J.	73	Dyspnœa, rapid pulse	Resection—enucleation left	Large solid compound adenoma; weight about 7 ozs.	R.	Drainage several days, primary union	100·4°	4	Transverse	Chloroform	May, 1907. Quite well. (Personal observation.)
339	June 18	Annie T.	33	Dyspnœa	Enucleation right, evidement left	Three solid adenomata, with blood extravasations, largest, size of big plum	R.	Drainage 1 day, primary union	100·6°	1	Transverse	Chloroform	May, 1907. Quite well. (Personal observation.)
340	June 18	Minnie S.	31	Deformity	Resection—enucleation left	Cystic adenoma, size of walnut	R.	No drainage, primary union	99	—	Transverse	Chloroform	February, 1907. Quite well. (Personal observation.)
341	June 25	Flora B.	36	Dyspnœa	Enucleation right	Cystic adenoma, size of walnut	R.	No drainage, primary union	99·2°	—	Transverse	Chloroform	April, 1907. Quite well. (Personal observation.)
342	July 2	Susannah B.	47	Dyspnœa	Resection—enucleation left; resection—extirpation right	Adenoparenchymatous; weight, right 2 ozs., left 5 ozs.	R.	Drainage 3 days, primary union	100·6°	3	Transverse	Chloroform	Nov., 1906. Quite well. (Letter.)
343	July 9	Alice T.	27	Dyspnœa	Resection—extirpation right	Parenchymatous, with multiple small adenomata, size of large walnut; weight $1\frac{1}{4}$ ozs.	R.	Drainage 2 days, primary union	100·2°	1	Transverse	Chloroform	October, 1906. Quite well. (Letter.)

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344	1906 July 9	Elizabeth L.	49	Dyspnœa	Resection—enucleation right	Solid adenomata, largest size of hen's egg	R.	Drainage 2 days, primary union	100°	1	Transverse	Chloroform	February, 1907. Quite well. (Dr. E. W. Cross, Leytonstone.)
345	July 16	Miss A.	40	Discomfort, slight dyspnœa	Resection—enucleation left	Solid adenoma, size of goose's egg, rather deeply seated behind clavicle	R.	Drainage 2 days, primary union	100·2°	1	Transverse	Chloroform	May, 1907. Quite well. (Letter.)
346	July 20	Ada W.	34	Dyspnœa	Enucleation right	Cystic adenoma, size of walnut	R.	No drainage, primary union	99·2°	—	Transverse	Chloroform	February, 1907. Quite well. (Dr. Scrase, Hampstead.)
347	Oct. 11	Mr. Arthur L.	52	Dyspnœa	Extirpation left	**Carcinoma,** attacking an old parenchymatous goitre; weight 5 ozs.	R.	Drainage 2 days, primary union. Patient went home on 9th day, quite well for the time	99·2°	—	Transverse	Chloroform	June 20, 1907. In good health and spirits; spends a large part of his time in shooting and going about his farm. (Personal observation.) Oct. 1907, dyspnœa and growth recurring. (Personal observation.)

348	Oct. 13	Mrs. A.	55	Slowly increasing dyspnœa	Resection—enucleation left	Three solid adenomata; weight 5¼ ozs., largest size of goose's egg, partly substernal	R.	Drainage 3 days, primary union	101°	3	Transverse	Chloroform	June, 1907. Quite well. (Personal observation.)
349	Oct. 18	Mrs. L.	30	Dyspnœa	Resection—enucleation left	Solid compound adenoma; weight 4 ozs.	R.	Drainage 1 day, primary union	99·4°	—	Transverse	Chloroform	June, 1907. Quite well. (Dr. Poulter.)
350	Oct. 22	Mary D.	41	Dyspnœa rapid pulse	Resection—enucleation left (2), enucleation right (3)	Five adenomata, mainly solid, from size of hen's egg downwards	R.	Drainage 2 days; small sinus formed a week after leaving hospital but soon healed	100·4°	2	Transverse	Chloroform	Sept., 1907. Quite well. (Letter.)
351	Nov. 1	Miss Ann B.A.	54	Dyspnœa	Resection—enucleation right	Solid adenoma, size of hen's egg, posterior part of lobe, also a small solid adenoma near isthmus, size of cherry	R.	Drainage 2 days, primary union; right vocal cord paralysed for 8 years (from pressure)	99·6°	—	Transverse	Chloroform	June, 1907. Quite well. Vocal cord in same condition as before operating. (Personal observation.)
352	Nov. 3	Emma R.	55	Dyspnœa	Resection—enucleation right	Cystic adenoma, size of hen's egg, ¼ fluid.	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	Sept., 1907. (Dr. J. R. Kingdon, King's Lynn.)
353	Nov. 12	Rhoda W.	30	Dyspnœa	Resection-enucleation right	Solid adenoma, size of small orange; weight 3 ozs.	R.	Drainage 2 days, primary union	101	1	Transverse	Chloroform	Sept. 23, 1907. Quite well. (Dr. W. A. Bowring.)
354	Nov. 15	Mrs L.	31	Deformity, discomfort	Enucleation right	Solid adenoma, size of hen's egg	R.	Drainage 1 day, primary union	100·2	1	Transverse	Chloroform	Sept., 1907. Quite well. (Dr. Calverley, Folkestone.)
355	Nov. 17	Amelia P.	27	Deformity, discomfort	Resection—enucleation right	Cystic adenoma, ½ solid, size of hen's egg.	R.	Drainage 2 days, primary union	100°	1	Transverse	Chloroform	January, 1907. Quite well. (Personal observation.)

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356	1906 Nov. 23	Mrs. R.	33	Increase in size, discomfort, going abroad	Multiple enucleation right	About a dozen small cystic adenomata	R.	Drainage 2 days, primary union	101°	2	Transverse	Chloroform	End of Dec., 1906. Quite well. (Dr. C. S. de Segundo.)
357	Nov. 26	Julia T.	38	Dyspnœa	Resection—enucleation right & left	Adeno-parenchymatous; weight 10 ozs.	R.	Drainage 3 days, primary union	100·4°	1	Transverse	Chloroform	Jan. 18, 1907. Quite well. (Personal observation.)
358	Nov. 27	Mr. M. F.	18	Discomfort, deep situation, increase in size	Resection—enucleation left	Solid adenoma, left lobe; partly substernal; weight 3 ozs.	R.	Drainage 4 weeks. Venous oozing, wound opened up and packed with gauze 19 hours after operation, secondary union, without suppuration, healed in 4 weeks	100·2°	4	Transverse	Chloroform	Feb. 19, 1907. Quite well. (Personal observation.)
359	Dec. 1	Mrs. Clement L.	24	Discomfort	Resection—enucleation left	Cystic adenoma, $\frac{1}{3}$ solid, size of hen's egg. Another solid adenoma, size of cherry	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	Sept., 1907. Quite well. (Dr. Surridge, Knutsford.)
360	Dec. 3	Babette G.	34	Severe dyspnœa	Resection—enucleation right	Solid adenoma in parenchymatous goitre; weight 5 ozs.	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	Sept., 1907. Quite well. (Personal observation.)
361	Dec. 3	Eliz'b'th P.	48	Dyspnœa	Resection—enucleation left	Cystic adenoma, with blood extravasion, and old fibrosis, size of orange	R.	Drainage 1 day, primary union	99·2°	—	Transverse	Chloroform	June, 1907. Quite well. (Personal observation.)
362	Dec. 10	Alice B.	37	Dyspnœa	Resection—enucleation right	Solid adenoma; weight $1\frac{1}{2}$ ozs.. partly substernal	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	Sep. 20, 1907. Quite well. (Letter.)

363	Dec. 18	Mrs. D.	68	Dyspnœa	Resection—enucleation right	Cystic substernal, with old hæmorrhage; weight 2 ozs.	D.	Drainage in very bad condition before operation; had also heart disease (mitral). Died of cardiac syncope soon after operation	—	—	Transverse	Chloroform (ʒij only)	
364	Dec. 20	Arthur J.	29	Dyspnœa	Resection—enucleation left	Cystic adenoma, size of orange	R.	Drainage 3 days, primary union	98·6	—	Transverse	Chloroform	Sept., 1907. Quite well when last seen. Gone to Canada. (Dr. Kent, Bexhill.)
365	1907 Jan. 1	Mrs. H.	49	Severe dyspnœa	Resection—enucleation left	Solid compound adenoma, largely intrathoracic; weight 5 ozs., vertical measurement 13 cm. (5 ins.) of which 6 cm. intrathoracic	R.	Drainage 2 days, primary union	100·2	1	Transverse	Chloroform	Sept., 1907. Quite well. (Dr. J. P. Roughton, Kettering.)
366	Jan. 7	Mary Jane R.	44	Dyspnœa	Resection—extirpation right	Adeno-parenchymatous; weight 6½ ozs.	R.	Drainage 2 days, primary union	100	1	Transverse	Chloroform	February, 1907. Quite well. (Personal observation.)
367	Jan. 7	Annie W.	18	Dyspnœa	Resection—extirpation right, partial extirpation left	Parenchymatous; weight, right 7 ozs., left 4½ ozs.	R.	Drainage 2 days, primary union	99·4	—	Transverse	Chloroform	May, 1907. Quite well. (Personal observation.)
368	Jan. 14	Sarah S.	43	Dyspnœa	Resection—extirpation left and half right	Parenchymatous; weight 14¾ ozs.	R.	Drainage 2 days, primary union	100·4	2	Transverse	Chloroform	July 23, 1907. Quite well. (Letter.)
369	Jan. 14	Walter B.	19	Dyspnœa	Resection—extirpation right	Parenchymatous; weight 4½ ozs.	R.	Drainage 1 day, primary union	101	2	Transverse	Chloroform	Sept., 1907. Quite well. (Personal observation.)
370	Jan. 28	Louisa K.	45	Discomfort, pressure	Resection—enucleation left	Cystic adenoma, size of orange, ¾ fluid	R.	Drainage 1 day, primary union	100·2	1	Transverse	Chloroform	June, 1907. Quite well. (Personal observation.)

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371	1907 Feb. 4	Marian T.	21	Deformity, slight dyspnœa	Resection—enucleation right	Cystic adenoma, size of an orange, weight 5 ozs.	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	Sept. 15, 1907. Quite well. (Letter.)
372	Mar. 4	Mabel B.	16	Dyspnœa	Resection—extirpation right and left	Parenchymatous, right 6½ ozs., left 5½ ozs.	R.	Drainage 2 days, primary union	99°	—	Transverse	Chloroform	June 3, 1907. Quite well. (Dr. Duncan, Croydon.)
373	Mar. 11	Annie B.	36	Dyspnœa	Resection—enucleation left	Solid adenoma; weight 3 ozs.	R.	Drainage 2 days, and again 7th and 9th day, for slight superficial suppuration under scar, otherwise primary union	100·6°	2	Transverse	Chloroform	April 2, 1907. Quite well on leaving hospital. Not seen since.
374	Mar. 18	Alice M.	34	Dyspnœa	Resection—extirpation right	Solid adenoma, size of small hen's egg; weight 1½ ozs.	R.	Drainage 3 days, primary union	100°	1	Transverse	Chloroform	July, 1907. Quite well. (Personal observation.)
375	May 13	Lizzie B.	31	Dyspnœa	Enucleation right	Cystic adenoma, size of Tangerine orange	R.	Drainage 2 days, primary union	99·6°	—	Transverse	Chloroform	Sept., 1907. Quite well. (Letter.)
376	May 24	Eliza O.	43	Dyspnœa	Resection—enucleation left	Compound adenoma; weight 8 ozs.	R.	Drainage 2 days, primary union	99·8°	—	Transverse	Chloroform	Sept., 1907. Quite well. (Personal observation.)
377	May 27	Rachel B.	49	Dyspnœa	Resection—enucleation left	Compound adenoma, weight 3 ozs.	R.	Drainage 2 days, primary union	99·6°	—	Transverse	Chloroform	Sept. 20, 1907. Quite well. (Dr. Tolputt.)
378	May 27	Edith S	29	Dyspnœa	Enucleation right	Cystic adenoma, size of walnut, inner and back part of gland	R.	No drainage, primary union	99·8°	—	Transverse	Chloroform	Sept., 1907. Quite well. (Dr. Burnes, Edmonton.)

379	May 29	Mrs. Amy B.	39	Dyspnœa	Resection—enucleation left	Solid adenoma; weight $3\frac{1}{2}$ ozs.	R.	Drainage 2 days, primary union	101°	3	Transverse	Chloroform	June 24, 1907. Quite well. (Personal observation).
380	June 3	Emma R.	19	Deformity, slight dyspnœa	Resection—enucleation right	Pure cyst, with clear fluid, size of an orange	R.	Drainage 2 days, primary union	99·2°	—	Transverse	Chloroform	July 29, 1907. Quite well. (Dr. E. Henry, Long Sutton, Lincs.)
381	June 4	Miss R. P.	35	Deformity	Enucleation right	Cystic adenoma, size of walnut, with old blood	R.	Drainage 1 day, primary union	99·6°	—	Transverse	Chloroform	July 29, 1907. Quite well. (Personal observation.)
382	June 5	Miss L. C.	49	Dyspnœa	Enucleation left	Cystic adenoma, mainly fluid, size of duck's egg	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	Sept. 6, 1907. Quite well. (Personal observation.)
383	June 10	Lily H.	19	Discomfort, slight dyspnœa	Resection—extirpation right	Adeno-parenchymatous, size of horse-chestnut, back and inner part of right lobe	R.	Drainage 2 days, primary union	99°	—	Transverse	Chloroform	October, 1907. Quite well. (Dr. Tremlett Wills, Farnborough, Hants.)
384	June 10	Charles B.	61	Malignancy	Extirpation right	Size of small orange, partly substernal	R.	Drainage 8 days, primary union, except in track of drain, where small sinus persisted for about 3 weeks and then healed	100·2°	4	Transverse	Chloroform	July 29, 1907. Doing well and able to do his work as farm labourer; scar quite firm and healthy, no pain, no sign externally of recurrence. (Dr. E. Henry. Long Sutton,) Oct. 11, 1907. Said to be still well and at work. (Dr. Henry.)

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385	1907 June 15	Mrs. G.	50	Dyspnœa	Resection—enucleation right and left	Substernal cystic adenoma, size of horse-chestnut left, 3 small solid adenomata right	R.	Drainage 2 days, primary union	99·8°	—	Transverse	Chloroform	Aug. 28, 1907. Quite well. (Dr. A. L. Davies, Wisbech.)
386	June 18	Miss Lucy M.	48	Discomfort, slight dyspnœa	Resection—enucleation right	Solid adenoma, size of horse-chestnut, deeply seated	R.	Drainage 1 day, primary union	99°	—	Transverse	Chloroform	July 11, 1907. Quite well. (Personal observation.)
387	June 21	Edith May E.	30	Dyspnœa, dysphonia	Resection—enucleation right	Soft solid adenoma, size of an orange; weight 2 ozs.	R.	Drainage 2 days, primary union	98·8°	—	Transverse	Chloroform	Aug. 10, 1907. Quite well. (Personal observation.)
388	June 25	Miss W.	40	Deformity, discomfort	Resection—enucleation right	Cystic adenoma, size of large orange, mainly solid; weight $5\frac{1}{2}$ ozs.	R.	Drainage 2 days, primary union	100	2	Transverse	Chloroform	Sept. 30, 1907. Quite well. (Personal observation.)
389	July 1	Caroline G.	30	Deformity, slight dyspnœa	Resection—enucleation left	Cystic adenoma, size of hen's egg	R.	Drainage 2 days, primary union	99·2°	—	Transverse	Chloroform	October, 1907. Quite well. (Dr. C. E. Tanner, Farnham.)
390	July 8	Mary Ann J.	51	Dyspnœa	Resection—enucleation right	Main tumour, cystic adenoma, size of orange, half solid, also substernal solid adenoma, size of walnut	R.	Drainage 2 days, primary union	99·2°	—	Transverse	Chloroform	Sept. 20, 1907. Quite well. (Letter.)
391	July 15	Ada L.	24	Deformity, slight dyspnœa	Resection—enucleation right	Cystic adenoma, size hen's egg, $\frac{5}{6}$ solid	R.	Drainage 1 day, primary union	99·8°	—	Transverse	Chloroform	Sept., 1907. Quite well. (Personal observation.)

392	July 16	Mrs. W.	33	Deformity, slight dyspnœa	Resection—enucleation right	Cystic adenoma, size of orange, ½ solid	R.	Drainage 1 day, primary union	99·6°	—	Transverse	Chloroform	July 29, 1907. Quite well. (Personal observation.)
393	July 22	Annie B.	43	Dyspnœa	Resection—enucleation left	Adeno-parenchymatous, size of orange	R.	Drainage 2 days, primary union	99·2°	—	Transverse	Chloroform	Sept. 20, 1907. Quite well. (Letter.)
394	July 22	John W.	18	Deformity	Enucleation and evidement, both right	Cystic adenoma, size of Tangerine orange, right upper horn, solid adenoma rather smaller, right lobe	R.	Drainage 1 day, primary union. (One previous operation elsewhere)	—	—	Transverse	Chloroform	Sept., 1907. "At work and apparently all right." (Dr. Fergusson, Painswick.)
395	Sept. 6	Rose A.	35	Dyspnœa	Resection—enucleation right	Solid adenoma; weight 6½ ozs.	R.	Drainage 2 days, primary union	100°	1	Transverse	Chloroform	Sept. 27, 1907. Quite well. (Personal observation.)
396	Sept. 9	H'nri'tta W.	38	Dyspnœa	Resection—enucleation left	Solid adenoma, 12 × 10 × 6 inches; weight 13½ ozs.	R.	Drainage 2 days, primary union	99·8°	—	Transverse	Chloroform	October, 1907. Quite well. (Dr. Durran, Leighton Buzzard.)
397	Sept. 9	Nellie H.	32	Deformity, slight dyspnœa	Resection—enucleation left	Solid adenoma, 11 × 8 × 4 inches; weight 8½ ozs.	R.	Drainage 2 days, primary union	99·8°	—	Transverse	Chloroform	Sept. 27, 1907. Quite well. (Personal observation.)
398	Sept. 10	Miss M.	37	Deformity, slight dyspnœa	Resection—enucleation right and left	Solid adenomata, right, size of duck's egg; left, 3 tumours, size of large cherries; total weight 3½ ozs.	R.	Drainage 1 day, primary union	100	2	Transverse	Chloroform	Sept. 30, 1907. Quite well. (Personal observation.)
399	Sept. 16	Ethel H.	25	Dyspnœa	Resection enucleation right	Adeno-parenchymatous; weight 4 ozs.	R.	Drainage 2 days, primary union	100·4	1	Transverse	Chloroform	October, 1907. Quite well. (Sister.)
400	Sept. 16	Mary B.	12	Dyspnœa	Resection—extirpation right, and left	Parenchymatous; weight 6 ozs.	R.	Drainage 2 days, primary union	99·4		Transverse	Chloroform	Sept. 27, 1907. Quite well. (Personal observation.)

DISCUSSION.

The PRESIDENT, in the name of the Section, accorded to Mr. Berry their hearty thanks, for he considered that the Section might be congratulated on having, at its first ordinary meeting, a paper of such very great interest brought before it. If those present looked round at the illustrations upon the walls, and still more if they looked at the patients in an adjoining room, it would be apparent that Mr. Berry had taken enormous trouble to bring a most interesting subject before them. He was sure all would appreciate the very great industry involved and the admirable character of the resulting work. There were many points of interest in the paper. The author had laid stress upon severe dyspnœa as a reason for operating upon a goitre. Another point was that dyspnœa depended upon direct pressure on the trachea, not upon disturbance of the recurrent laryngeal nerve. He remembered Mr. Berry's admirable specimens showing the pressure effects of goitre upon the trachea, and the softening of its walls, with narrowing of the lumen. He had been much struck by the almost invisible character of the scar left after operation. The incision which Mr. Berry adopted was the right one, not only for convenience of operating, but because of the appearance after operation. He would like to hear from the author why he had excluded ether from the anæsthetics he employed for such cases. Several of the very small number of deaths which ensued were due to cardiac failure soon after the operation, and it occurred to him that if a more stimulant anæsthetic had been used the number of deaths might have been even smaller. He only asked for information, feeling sure that Mr. Berry had good reason for his preference.

Sir VICTOR HORSLEY said he assumed that everybody now used Mr. Berry's method of operating; he certainly had for many years, draining for twenty-four hours, and bringing about immediate union in the same way. He believed that Professor Kocher did not now drain in most cases. For himself, he regarded drainage as very important. He had never allowed the anæsthetist to give ether in cases of goitre for the last ten years because such patients secreted mucus much more freely in the trachea than ordinary cases, and ether facilitated the secretion of mucus more than did chloroform. The embarrassment of respiration, of which much had been made, was proportional to the dose of anæsthetic. If only 0·5 per cent. strength were given with the chloroform inhaler, no trouble was caused. If dyspnœa occurred on the table, as a rule it was because the anæsthetist was giving too much chloroform or using a mucus-producing anæsthetic. There were innumerable points of interest in the paper, but he proposed to touch on only one or two concerning the pathology, because the decision as to operation must depend on the views as to pathology. First, with regard to parenchymatous goitre, the whole question of operation or not seemed to depend on the degree of athyroidism. There was a certain number of young subjects, under 21 years of age—he thought 21 was low for the average age—on whom one would never think of operating, because under thyroidal treatment they got well without operation; but again and again one had been asked to see adult patients of higher age on whom the thyroidal treatment had been employed. Those patients could not be cured by such means, they had gone past the period of growth, and they must be operated on; but young subjects, certainly those under 21, should be treated most conscientiously with thyroid before the question of operation was dreamed of. All must agree with Mr. Berry's description of the adenoma and as to it being practically the only cause of a thyroid cyst; but there was another question with regard to the pathology of adenomata which had scarcely received sufficient attention, especially in regard to the posterior tumours, such as were illustrated by one of the diagrams. He had previously shown that these posterior tumours were often pure parathyroidal adenomata. He had operated in Queen Square Hospital on a parallel case last year. The patient had not only an intrathoracic calcified growth, but also a large parathyroidal adenoma level with the hyoid. He diagnosed the upper tumour to be an accessory thyroid, but microscopically it was proved to be a parathyroidal adenoma. When he was surgical registrar at University College Hospital nothing was known of parathyroids, but accessory thyroids were described; and he now believed many such tumours were parathyroidal. These tumours must be approached in the way which Mr. Berry had so eloquently described, in order to avoid unnecessary involvement of the recurrent laryngeal; but he did not suppose any surgeon present now saw trouble with the recurrent laryngeal nerve; that was now ancient history, because the methods of operating were now so thoroughly understood. Lastly, he desired to draw attention to the fact that not only in regard to the *formes frustes* of Grave's disease, but in ordinary simple adenoma, if one investigated the condition of the patient after the operation, one would find they reported the disappearance of numerous neurasthenic symptoms. Indeed, although they had been going about apparently in perfect health, and very often doing their work, they had been more or less neurasthenic from the toxic condition due to perverted secretion produced by the adenoma. By removing the adenoma one also removed the source of the toxic symptoms. Lastly, the thyroid gland was certainly very sensitive to physiological venous pressure; and although Mr. Berry would not be able to prevent his patients singing his praises, he did not think they should sing them to music.

Mr. W. G. SPENCER congratulated Mr. Berry on the very distinguished selection of cases and specimens which he had shown. His own memory went back to the time when Mr. Berry, in the post-mortem room at St. Bartholomew's Hospital, was continually dissecting out thyroids and beginning the study of the subject. It was a subject upon which he had continually learnt from Mr. Berry, and in regard to which one had simply in a large measure to follow him. His own small experience had been largely due to the assistance which Mr. Berry had always been ready to give to his contemporaries. It was of great importance that superficial chloroform anæsthesia was all that was necessary, especially in contrast with the dicta of Professor Kocher and others, because it must save a great number of such patients from the mental shock, which must correspond largely to that following after accidents. If he mentioned one or more points it was because Mr. Berry, at the end of his paper, stated that some further information concerning exophthalmic goitre might be valuable. It seemed to him that, quite apart from those forms of exophthalmic goitre in which the thyroid was enlarged, and in which symptoms must be due to dyspnœa, there were cases without enlargement, especially suffering from extreme rapidity of pulse and many other nervous symptoms, whom he thought at any rate a small operation might benefit compared with other treatment. He referred to dividing the isthmus, or taking away a little of the thyroid gland on each side, such a limited operation as such patients could stand. The only case which, in his own experience, had died was one whose specimen was in the Westminster Hospital Museum. She came in practically unconscious, and she was set up on the table without an anæsthetic, and after dividing the thyroid isthmus he had to put a tube in the trachea; it was found post-mortem—it was a condition that Mr. Berry had not mentioned—that she died because of the enormous thymus which she had in addition. In another case the patient had been unable to lie down, and there were other symptoms, such as crying fits. The pulse was 130, although she had been in bed some time. The isthmus was divided and a small portion of it was removed. She said she was considerably relieved, and life became more tolerable. She must have been an exophthalmic case of some form, because a year afterwards she came back with severe pain on one side which was referred to the superior sympathetic ganglion. He excised that, and it relieved her pain on one side. She came back six months afterwards with an identical pain on the opposite side, and that led to the excision of the opposite superior cervical ganglion. She was now in a more comfortable condition, although not well; her eyes were prominent, she had, when excited, a rapid pulse, and was anæmic. Another case was that in which the goitre shrank and became fibrous. Then it constricted the trachea, and in a different way from other cases he had seen; it seemed as if there were some annular stricture. He cut away a portion of that dense thyroid and divided the trachea down, almost in the same fashion as one would divide a urethral stricture. Although she had afterwards some nervous symptoms, she was in a reasonable state of health years afterwards. There were also other cases of exophthalmic goitre in which division of the isthmus seemed to do good, although he did no think they were cured. He thought the benefit was greater than it would have been by medical treatment. Mr. Berry mentioned that many cases need not be operated upon, but that applied also to thyroid tumours above the thyroid, those which ran up into the tongue. Some surgeons at present excised those tumours, which were probably commoner abroad than here. Such patients had been shown to have practically no thryoid gland in the proper position at all, merely a mass of fibrous tissue, and the result of such extensive operations, which were quite unnecessary, was to produce a condition of myxœdema. But he thought Mr. Berry would agree that there were cases in which he might be a little more insistent with regard to the necessity of operations. Sir Victor Horsley had mentioned cases later in life—he would leave Mr. Berry to state the exact age—but after about middle life surely every enlargement of a thyroid gland in the nature of a tumour must be looked upon from a surgical point of view, because so many proved to be malignant. He admitted that some might be gummatous, and some might be simply benign adenomata, but so many might possibly be sarcomata and carcinomata, which, if excised immediately, might give better results than those malignant cases which Mr. Berry had dealt with. For instance, there were spindle-celled sarcomata, and cysto-carcinomata and adeno-carcinomata which began in the centre of the gland. Possibly those might be cured by early excision, though he did not say there was much chance for a true scirrhous carcinoma affecting the whole gland. If all the cases after a certain age were presumed to be surgical, and not treated medically—and Sir Victor Horsley had said it was of little use to treat such medically—there would be better results for malignant disease in that region.

Mr. Herbert Paterson said that the question of the anæsthesia for the cases under discussion was an important one. Sir Victor Horsley had supported chloroform, and if he himself had spoken on the subject two or three months ago, he would have done the same. But recently, during a visit to America, he was introduced to the plan of giving ether by the open method, and was much struck by the simplicity and the ease with which it was given for all kinds of operation, but in particular for difficult thyroid operations. Sir Victor Horsley had mentioned that ether was apt to increase the secretion of mucus, undoubtedly a great disadvantage. But he had seen ten operations of this kind done in America under ether, and there seemed to be no extra secretion of mucus nor any inconvenience to the operator. Recently he had operated on one of these cases under ether given by the open method by Mr. Bellamy Gardner, and he had been much gratified by the quietness and excellence of the anæsthesia. He thought it ought to be considered whether in this country the giving of ether by the open method should not be advocated. He had himself seen two thyroid cases die on the operating table from the anæsthetic, and he believed such a calamity would be prevented by ether given by the open method. While he was in Atlantic City there was a symposium between the medical and surgical sections on operation for exophthalmic goitre, and he heard the leading physicians and surgeons in America give their opinions. In America they looked with great favour on the surgical treatment of exophthalmic goitre by extirpation of one half of the gland. During a fortnight's stay at Rochester he saw Dr. Charles Mayo perform four or five of those operations for goitre, and they were all genuine cases of the disease, because they all had exophthalmos. He did not regard the cases mentioned by Mr. Spencer with rapid pulse and tremulousness as true examples of the disease. Those cases which he saw treated surgically in America did remarkably well. Nearly all of them were up on the second or third day, and one of them was up witnessing other similar operations two days later. He made observations on the patients afterwards, and found them much benefited by the operation. As Mr. Berry said, it was desirable to know the late results of the cases, and, so far as he knew, no one in America had published accurate statistics as to the remote result of their cases. At the symposium, Dr. Mayo said he had performed 110 operations for exophthalmic goitre, with only nine deaths, or 8 per cent. mortality. He also stated that 50 per cent. of those patients had been permanently relieved, 25 per cent. partially relieved, and in the remaining 25 per cent. the operation had not been completely satisfactory, although some of the symptoms were relieved. Dr. Mayo laid stress on the fact that while the nervous symptoms rapidly disappeared, the exophthalmos sometimes remained for some months after the operation.

Mr. BERRY, in reply, said other speakers had really answered the President's question as to the reason for the preference of chloroform as the anæsthetic. Still, he thought ether administration by the open method deserved a trial, and he proposed to use it in his next few cases. In answer to Sir Victor Horsley's point, the average age of the parenchymatous goitre cases was really about 18; if three or four exceptional cases over 30 years of age be excluded, it was between 14 and 18 years of age that parenchymatous goitre was so apt to be dangerous, and many cases at this age would not yield to thyroid treatment; he never operated upon such cases without having first tried medicinal measures and satisfied himself that something more must be done. Sir Victor Horsley's remarks about parathyroids were very interesting, but he, Mr. Berry, was not very clear about the pathology or the surgery of parathyroids. He thought there was very little in the talk about parathyroids. In any case, it was somewhat foreign to the subject of his paper. He was glad that Sir Victor Horsley agreed with him that most cases of adenomata were relieved not only of the dyspnœa, but also of other symptoms. He must not allow Mr. Spencer to draw him into a discussion on exophthalmic goitre. He only mentioned the matter because he had not excluded all the malignant cases and the exophthalmic ones, which some surgeons did when giving lists of cases of thyroid disease. Mr. Spencer's remark about removing a small quantity of thyroid tissue was in opposition to Professor Kocher's teaching, which was that the safer course was to remove a large quantity. He must leave Mr. Spencer and Professor Kocher to argue that out between them. He was not much disposed to do goitre operations on elderly people when they had no dyspnœa. He only operated on them when there was dyspnœa or a reasonable suspicion of malignancy, and those cases were not very numerous. But it was common for people over 40 years of age to be sent to him with small hard tumours, thought to be malignant, which he did not hesitate to pronounce to be simple cysts of the thyroid. Such cases were extremely common. Mr. Paterson's remarks were very interesting, and he had seen him remove a thyroid tumour under ether given by the American method. The chief objection seemed to be that it required an enormous amount of ether, and the anæsthetisation was rather a slow process. In reference to the American series of 110 cases which Mr. Paterson mentioned, he asked whether they were all genuine cases, or were many of them *formes frustes?*

Surgical Section.

December 10, 1907.

Mr. J. Warrington Haward, President of the Section, in the Chair.

A Case of Complete Gastrectomy.

By B. G. A. Moynihan, M.S.

The following are the notes of a case in which I removed the whole stomach on account of its universal implication in a malignant growth. For the notes, and for his care of the patient after operation, I am indebted to Mr. E. R. Flint, house surgeon; the pathological investigation and report have been most kindly undertaken by Dr. Craven Moore.

The patient, M. L., a man aged 43, was sent by Dr. Peter Macdonald Acomb and admitted to the Leeds General Infirmary on May 24, 1907. He is married, has two healthy children, and has always been healthy himself, the only illnesses he can remember being influenza two or three times, and an ischio-rectal abscess some ten years ago. He has always had as his occupation the management of horses, and has been in his present situation as coachman for five years. He says that all his relatives, so far as he can remember, have died of old age. The patient was in good health up to two years ago, when he gradually began "to go off his food." By this he means that he did not want his food quite as he had been accustomed to do. He could eat anything, but was afraid of the pain which he knew would come on after taking food. This pain, which was relieved by food for an hour or so, was situated in the epigastrium, and continued until he vomited, when it at once disappeared until an hour after his next meal. The kind of food made no difference to the pain. The vomiting commenced as a profuse gush, and in the vomit he recognised food which he had taken at his last meal. There was never to his knowledge any hæmatemesis.

After the first six months he was free for some weeks from pain and vomiting, but at this time he was under medical supervision, and was taking liquids chiefly; he had his stomach washed out every other day for two weeks, and was away from work for eight weeks. The stomach contents at this time were analysed, as there was a suspicion in the doctor's mind of cancer, but the analysis gave a normal result. After eight weeks he returned to work, still taking fluids, and felt much better, but every now and again he had attacks of vomiting and pain; he noticed that his pain gradually began to appear at shorter intervals after his food, until it eventually came on as soon as food was taken, but was still relieved by vomiting. All this time he was steadily losing weight, falling from 11 st. 4 lb. to less than 9 st. He had also noticed that the amount of fluid taken at any one period was much less than formerly, until last Christmas the amount was so diminished that he was only able to take three or four mouthfuls at a time before he experienced a feeling of discomfort and vomited. From that time up to his admission this diminution in amount continued.

On admission the patient was in fairly good condition, but looked as though he had been a stouter man at some time; his skin was somewhat loose, his muscles flabby, and his cheeks a little hollowed. Weight, 8 st. 2 lb. When he was given some fluid to drink he merely sipped it a mouthful at a time, and had to wait a few seconds (about ten seconds) before the fluid "settled"; he gulped and strained his neck forward as though trying to get the liquid to pass an obstruction. After a few seconds he would appear comfortable and ready for another mouthful. He still vomited occasionally: the vomit was not fermented nor sour. He could not take any solid food at all, with the exception of cheese, a few "nibbles" of which he enjoyed. He never had any desire for food; in all about 30 to 40 oz. of fluid were taken daily.

Abdominal Examination.—The abdomen looked thin and excavated; its anterior surface receded sharply from the raised costal margin. Palpation revealed nothing abnormal; the walls were rigid, and nothing unusual could be felt through them. Two very small doses of tartaric acid and carbonate of soda were given separately. Instantly some foam gushed out of the mouth. On examining the abdomen a puffy swelling about the size of a billiard ball was seen and felt in the left upper half of the epigastrium, immediately below the costal margin. No other part of the stomach was distended, and the inflated portion was everywhere quite definitely circumscribed. The conclusion reached from this examination was that there was possibly an hour-glass stomach, with a very small cardiac complement, and that this was due to the cicatricial contraction of an ulcer on the lesser curvature close to the cardia. A stomach-tube was then passed. It went 17 in. quite readily, but not further than that. Only a small quantity of fluid could be introduced. A little over 4 oz. was the most that could be retained; when this had flowed into the stomach the patient began to complain of pain, great tightness and oppression, and was obviously greatly distressed. The fluid soon returned clear; on one occasion a little blood was seen. Bile was frequently seen in the washings. At one examination it was found that when a measured quantity of fluid was used for the washing, only two-thirds of it was returned through the tube. On two subsequent occasions the stomach was inflated: on one the swelling at the cardiac end was again well seen; on another it could not be demonstrated.

The following was the result of the examination of a test meal:

Macroscopic ...	Food badly digested. Blood.
Chemical ...	Blood. No free HCl. No lactic acid.
Microscopic ...	Yeast present. Sarcinæ present. *B. geniculatus* present. Other organisms were numerous. Pus abundant.

The diagnosis was made of hour-glass stomach with a very small cardiac complement. Owing to the impossibility of inflating the distal or pyloric part of the stomach reference was made, when the case was demonstrated to the ward class, to the possibility of its being an example of "leather bottle" stomach, the "linitis plastica" of Brinton. Operation was advised and was performed on May 31, 1907, in the following manner: The abdomen was opened in the middle line by an incision which at first was about 3 in. in length, sufficient to allow of exploration, but which was increased subsequently to a length of 8 in. At the outset there was a very serious difficulty in exposing the stomach. The patient was a man who had been stout, but who had lost weight rapidly; the anterior abdominal wall therefore shelved downwards from the elevated costal margin in such manner as to make the upper part of the stomach appear to be at great depth from the surface. The patient, moreover, was not at all comfortable under the anæsthetic, and I had to wait a long time after opening the abdomen before I could proceed with the operation. When the stomach was exposed it was seen to be small in size, with walls of great thickness and solidity. The whole organ, indeed, felt solid, resembling a very large uterus, having thick walls and an insignificant cavity within it. The surface was smooth, white, opaque; there were no adhesions, and but few obviously enlarged glands along the curvatures. Towards the cardiac end the stomach was larger than elsewhere, so that the organ had something of the shape of a Florence flask; the larger part, however, was still very much smaller than the normal.

This being the condition of the stomach it was at once evident that the performance of gastro-enterostomy was impossible, for there was no sufficient cavity in the stomach to admit of any anastomosis being made. The alternative procedures were complete gastrectomy, and jejunostomy or duodenostomy; after some deliberation I decided in favour of the former, and I proceeded at once to remove the whole stomach.

It was at this point that the abdominal incision was enlarged. Hot moist swabs in two layers were then packed into the abdomen in the usual manner to isolate the field of operation. The stomach was now depressed as far as possible by forcible traction made by an assistant, and two long clips applied to the coronary artery at its origin from the cœliac axis. The artery was divided between the clips, and its proximal end was ligatured. The upper and lower coronary group of glands was detached downwards towards the stomach by gauze stripping, and the cardiac end of the stomach denuded by the same means. The gastro-hepatic omentum was divided, after ligature, as close up to the liver as possible, until the upper border of the pylorus was reached. Here, by gauze stripping, the pyloric artery and the gastro-duodenal artery were exposed as they separately arose from the main hepatic trunk. The pyloric artery was ligatured and divided, and the finger then passed downwards behind the pylorus, and made to present at the lower border of the duodenum, where an opening was made in the great omentum. Through this opening the blade of a clamp was passed upwards, behind the duodenum, to present above the pylorus. When this clamp was closed it lay about 1 in. beyond the pylorus, and on the stomach side of it there lay the subpyloric group of glands. A second clamp, with rubber-covered blades, was now applied distal to it, and the duodenum was cut between them. A single strong catgut suture was then passed through the proximal part of the duodenum and round the clamp to prevent the clamp from slipping away. The distal end of the duodenum was then closed by a continuous catgut suture taking all the coats and by a double layer of Pagenstecher thread suture above this. The clamp holding the proximal part of the duodenum was now covered with a gauze swab, and was lifted well towards the left, exposing the gastro-duodenal artery more conspicuously. The artery was ligatured and divided. Along the whole length of the greater curvature the gastro-hepatic omentum was divided at a distance from the stomach of from 1 in. to 2 in., so that all glands, including one or two dropped glands, were left attached to the stomach.

The whole stomach was now free, for the gastro-hepatic omentum had been entirely divided; the duodenum was severed, and the gastro-colic omentum ligatured and cut free. The whole stomach hung pendulous from the œsophagus. At this point the anæsthetist was asked to fix the patient's neck as much as possible, in the hope that this might enable the œsophagus to be pulled down a little more readily, and it seemed that this hope was fulfilled. The œsophagus was dragged upon with a fair degree of force until at least ¾ in. of it was visible below the diaphragm.

The next step, and the most important and difficult of all, was the anastomosis of the œsophagus to the jejunum. The transverse meso-colon was already exposed on its upper surface in the wound; it was divided in an avascular area, and the upper loop of the duodenum pulled through it. A point on this about 8 in. from the duodeno-jejunal flexure was selected for the anastomosis. A piece of it about 2½ in. in length was laid transversely along a line immediately behind the œsophagus. As it lay there, transversely, the right leaf posterior, its upper end was to the left, its lower to the right. The anastomosis was now begun by introducing eight light interrupted sutures between this portion of the jejunum and the œsophagus. The part of the circumference of the jejunum used was that on the surface, which was now posterior, and on this surface about ¾ in. from the mesenteric attachment. As the sutures were introduced into the œsophagus this was made to present, and was well exposed by a forcible and continuous downward traction upon the stomach. The stomach, wrapped in a hot gauze swab, was used, and most efficiently used, as a retractor, or rather as an instrument of traction, upon the œsophagus. The help derived from this manœuvre was far greater than could be believed from a mere description. It converted what would have been an excessively difficult feat into one of comparatively easy accomplishment. Eight interrupted sutures then were introduced until the whole of the posterior half of the œsophagus was securely attached to the jejunum. In front of these a continuous suture was now introduced, exactly as in the operation of gastro-enterostomy from left to right; the needle carrying this suture was then laid aside to be presently resumed. The attachment of the œsophagus to the jejunum seemed now quite secure on this posterior aspect. In front of this continuous suture a small opening was made into the œsophagus and into the jejunum, at the extreme left end of this attachment. A continuous through-and-through Pagenstecher thread suture was now begun, and a few turns of the needle taken until the whole length of the small openings made had been united. These openings were then enlarged little by little from left to right, as they were enlarged their cut edges were sutured by the same continuous stitch. This sequence of a small incision, a few stitches, slight enlargement of the incision, a few more stitches, was continued until the whole of the posterior part of the œsophagus was divided and sutured to the incision in the jejunum. Around the anterior wall of the œsophagus the same sequence was continued, the stitch being now changed to the "loop on the mucosa" form (see "Abdominal Operations," second edition, page 395). The result was that the stomach was retained as a tractor, drawing down the œsophagus until the last piece was severed, and at that moment the line of anastomosis was almost complete. Finally the outer continuous suture previously laid aside was resumed, and continued round the anterior surface of the œsophagus and jejunum to its starting point, where it was tied and cut short.

The suture lines were now complete. There were, it will be seen, eight interrupted posterior sutures, intended as anchor sutures, and the two continuous sutures, as in the usual operation of gastro-enterostomy. A few anterior anchor sutures fixing the jejunum and œsophagus to the diaphragm were now taken, and the main part of the operation was now complete. The great omentum was turned upwards over the operation area and the abdomen closed.

The patient had borne the operation well. There had been no soiling of the operation field nor any exposure of viscera. As soon as the patient was put back to bed the continuous administration of saline fluid by the rectum was commenced. In the first twenty-four hours 9 pints were taken; in the second twenty-four hours 6 pints. After this it was discontinued. The help given by the absorption of 15 pints of normal saline solution within forty-eight hours is probably difficult to exaggerate. During this time, contrary to my usual practice, I gave no fluid by the mouth, but the patient was allowed to flush his mouth as often as he wished. He never complained of thirst, and did not suffer any great amount of pain. He was kept lying flat on his back, with the head propped well forward.

The administration of fluids by the mouth was begun very cautiously on the third day. Two teaspoonfuls of water were given every half-hour; on the fourth day this quantity was increased to 2 oz. every half-hour. On the fifth day 5 oz. were given hourly; water and peptonised milk and albumen water were given in succession. On the sixth day 2 pints of these fluids were taken while the day nurse was on duty, and 1 pint 6 oz. during the night. These quantities, of the same fluids, were slowly increased, until on the tenth day 5 pints were taken in twenty-four hours. On the eleventh day beef-tea and Benger's food were given;

on the fourteenth day milk pudding; on the eighteenth day bread and butter. During the third week the patient told us every day that he was hungry, a sensation he had not experienced so keenly for two years. At the end of the third week he began to take meals of fair quantity, consisting of minced chicken, milk puddings, &c. He was kept in bed for eighteen days, and on the twenty-second day was sent to a convalescent hospital. On leaving the hospital his weight was 8 st. 12 lb., a gain of 10 lb. On August 21 he weighed 10 st. and was able to eat all foods.

This is the second occasion upon which I have been called upon to perform complete gastrectomy.[1] The circumstances present in the two cases were similar; the stomach was small, with thickened walls and a cavity greatly reduced in size; it was invaded in every part by cancer, the glands were only slightly affected, there were few adhesions, no invasion of the parts around by the growth, and no secondary deposits. It has been computed by Fenwick[2] that 14 per cent. of all patients dying with carcinoma of the stomach show no extension of the disease beyond this organ. The type of cancer in both these patients was atrophic, the malignancy probably of a low grade. It would seem that conditions of the kind enumerated are essential to the successful carrying out of the operation of complete gastrectomy. In my first case, which proved fatal, I adopted a technique which I thought satisfactory. After the operation I gave much thought to the details of the procedure and endeavoured to construct a method which I should carry out if the opportunity again came to me. I had determined to make use of the stomach tube passed through the œsophagus into the jejunum as a sort of cylinder upon which to suture, and I considered that the fixation by a catgut suture of the tube to the cut end of both œsophagus and jejunum (the suture being, of course, buried by the continuous sutures along the line of anastomosis) would help to make the feeding of the patient during the time of healing of the wound a simple and a safe matter. But when I came to perform this second operation I realised, as I saw the stomach pendulous from the œsophagus, that it might be used with the very greatest advantage to hold the œsophagus in a fixed position until my suture lines were practically complete. I feel sure that this point is one which has solved the greatest of all difficulties in the operation of complete gastrectomy, and it embodies, moreover, a technical principle which is applicable to other operations than this.

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[1] *Brit. Med. Journ.*, 1903, ii., p. 1458.

[2] "Cancer of Stomach," p. 54.

PATHOLOGICAL REPORT.

By Dr. Craven Moore.

The specimen includes the whole stomach and about ¼ in. each of the œsophagus and duodenum. The stomach is greatly diminished in size, its length being 4¼ in.; in form it is tubular, gradually contracting towards the pylorus, and about the middle third it presents several deep transverse folds, which it is impossible to obliterate by tension; in consistency it is firm and elastic. Attached to the stomach are portions of the gastro-hepatic and the gastro-colic omenta, and in the former are several lymphatic glands, which are firm and of normal size; the omenta themselves appear quite normal. The serous coat of the stomach over the fundus and middle third of the organ appears somewhat thickened and more opaque than normal, and here and there presents small white nodules about a pin's head in size. The wall of the stomach is greatly thickened and indurated, the thickening being greater in the proximal two-thirds of the organ, where it measures ½ in., than in the pyloric portion, where it measures ¼ in. The cut surface of the wall shows a mucous layer rather thinner than normal, lying on a much thickened, greyish-white, dense, submucous coat, a well-developed muscular coat in

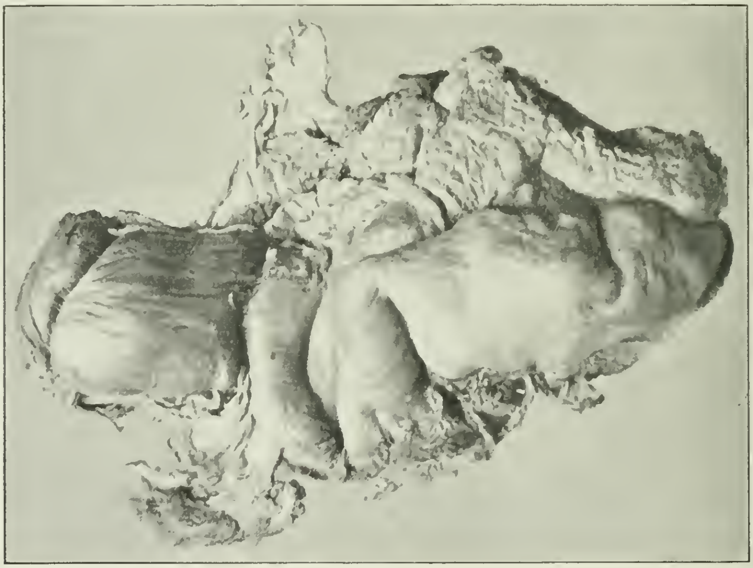

Fig. 1.
Stomach seen from the outside.

which the individual fasciculi are rendered more than usually evident in many places by an increase in the intermuscular connective tissue, a subserous and a serous coat, which also appear to be slightly thickened.

It is very obvious that the great thickness of the stomach wall is the result chiefly of the increased extent of the submucous coat. The cavity of the organ, greatly diminished in extent, is divided into two distinct loculi by a zone of contraction situated in the middle region of the stomach, where the cavity is reduced to a narrow passage ¼ in. in diameter; this zone of contraction corresponds to the deep folds marking the external surface of the organ. The cardiac loculus corresponding to the fundus has a diameter of 1¼ in., it is rounded in form and its mucous lining has a mamillated appearance. The pyloric loculus, of more tubular form, is divisible into two portions by the character of its mucosa: in the proximal portion, corresponding to middle third of the stomach, the

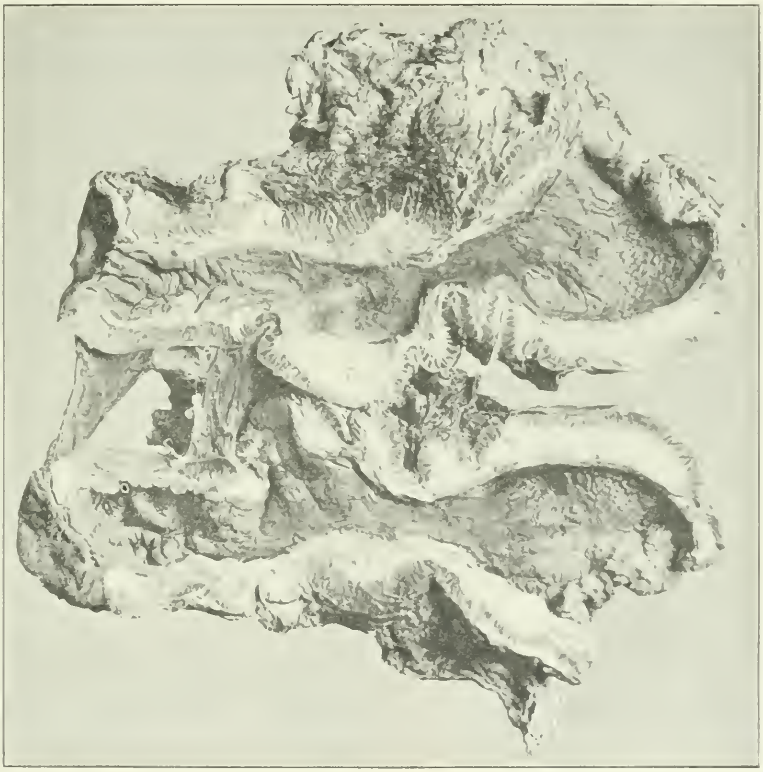

Fig. 2.
Stomach cut across.

mucous lining is smooth and thinner than normal; in the distal portion, which apparently corresponds to the pyloric antrum, the mucous lining is of normal thickness, and is thrown into a series of irregular folds; it is in this portion of the organ that the submucous coat shows the least change. The two orifices of the stomach show no indications of contraction, and the stomach wall is here of normal thickness.

Microscopic Examination.—Sections of the wall of the cardiac loculus show a thin mucous layer in which only the deeper parts of the tubules are present, and these present extensive and irregular proliferation of their cells, strands of which can be traced down into the underlying submucosa; between these proliferous elements, in which they appear to be imbedded, there is granulation tissue (plate, fig. 4.) The submucous coat consists of a dense white fibrous tissue in which the fasciculi of fibrils are well defined, run more or less parallel, have a wavy oblique course and resemble very closely the fibrous elements seen in dense fibromata and in the atrophic variety of scirrhous carcinomata. Towards the mucous layer this coat is sharply defined; on the other side it penetrates the muscular coat by a series of strands running between the muscular bundles (plate, fig. 5). This dense fibrous tissue shows a great paucity in cells, but in its innermost layers it shows here and there small collections and strands of epithelial cells, many of which can be seen to be directly continuous with similar cells in the mucosa,

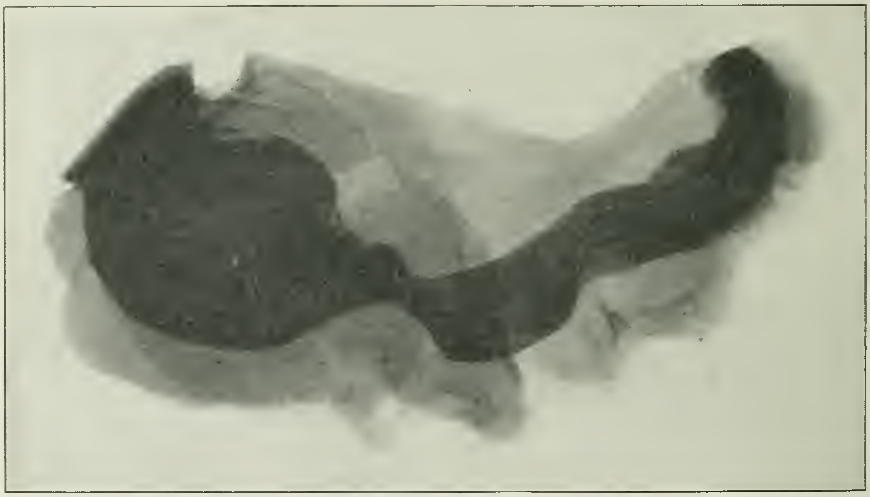

Fig. 3.
Stomach filled with bismuth solution and skiagraphed.

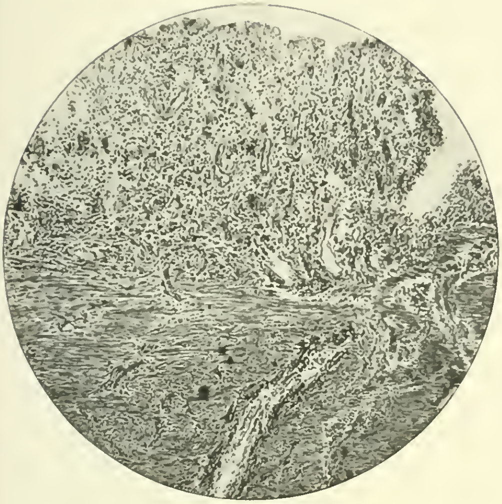

FIG. 4.

Section through the mucous coat and subjacent portion of the submucous coat, showing proliferous epithelial elements extending into latter.

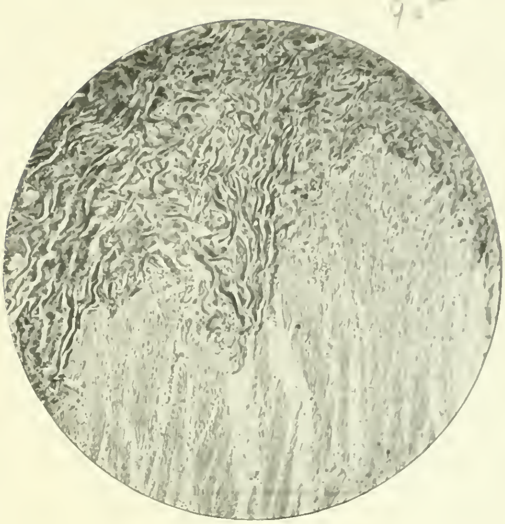

FIG. 5.

Section through adjacent portions of submucous and muscular coats, showing character of fibrous hyperplasia in former and its invasion of latter.

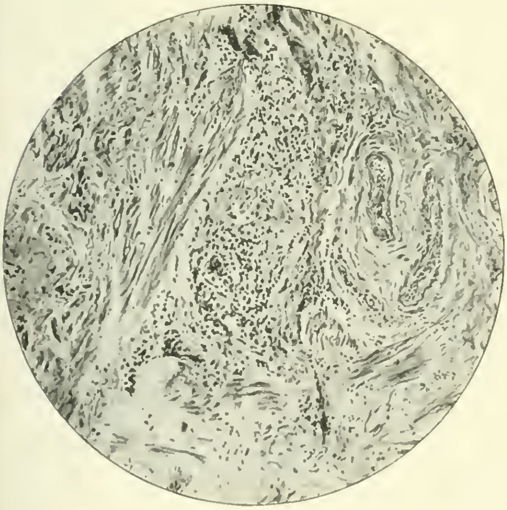

FIG. 6.

Section through muscular coat, showing an area in which there is a great new formation of fibrous tissue with proliferous epithelial cells, and atrophy of the muscle cells; also an artery showing periarteritis.

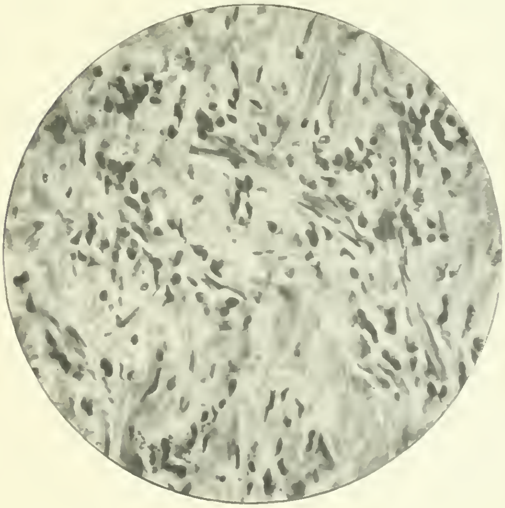

FIG. 7.

Section through the submucosa showing proliferous epithelial elements among the dense fibrous tissue

MOYNIHAN: A Case of Complete Gastrectomy.

and in the remainder of its extent, even where it penetrates between the bundles of the muscular coat, there are to be seen isolated strands and small islands of similar cells between the white fibres, the latter in particular being commonly adjacent to some blood-vessel and accompanied by a number of leucocytes (plate, figs. 6 and 7). The blood-vessels are few in number and show some thickening of their external coats, but no obvious change in their middle or internal coats. The muscular coat does not appear to be increased in thickness, the muscular bundles are separated by strands of dense fibrous tissue penetrating it from the submucosa, which become more and more attenuated in the outer layers and so disappear; the muscle-cells here and there show indications of hyaline transformation, but otherwise are normal. The subserous coat is slightly thickened, and very occasionally one sees a few epithelial-like cells. The serosa appeared normal. Sections taken from the zone of contraction present similar changes, the mucosa is even thinner, and the folding of the stomach wall is seen to be due to contraction of the fibrous submucosa.

Sections from the pyloric portion present appearances very little removed from those of the normal organ. Sections of the lymphatic glands from the gastro-hepatic omentum show here and there small collections of cells very similar to those described in the submucous coat, situated in the trabeculæ and in the perivascular lymphatics. Attempting to interpret these appearances, there can be no doubt that we have to deal with an extensive fibrous hyperplasia commencing in, and chiefly limited to, the submucous coat and involving that coat almost exclusively in its proximal two-thirds, a condition which so far corresponds with Brinton's conception of plastic linitis. The presence of the scanty proliferous epithelial elements in the remains of the mucosa and in the midst of the newly formed fibrous tissue of the submucosa demonstrates that the lesion is not merely an inflammatory one in this case, but that it is essentially a neoplastic change of a particular type, which has its analogue in the so-called atrophic scirrhus met with in the mammary gland.

Dr. CRAVEN MOORE gave an epidiascope demonstration of slides.

DISCUSSION.

The PRESIDENT (Mr. Warrington Haward) said he was sure the Fellows would agree that the paper they had listened to had been an extraordinarily interesting one. The operation described was most interesting, and the result was equally so. The patient was in the next room, and specimens and preparations of the stomach were on view. There were many points calling for discussion and consideration, though probably not many of those present would have had experience of a similar operation. The condition of the stomach was most curious, its contraction and the great thickening of its walls making it almost resemble in appearance a uterus. With regard to the diagnosis, the disease appeared to correspond with that described by Brinton as plastic linitis. A matter of great interest was the physiology and result of the removal of the stomach. In that connection nothing was more surprising to him than the healthy condition of the man from whom the stomach had been removed, and his capacity to eat even things which many anatomically complete people would regard as somewhat indigestible. Recently we had had opportunities of learning how people fared without a considerable portion of their large intestine, and there would perhaps now be an opportunity of seeing how well dyspeptic people could dispense with their stomachs. It would be interesting to hear what pathologists had to say on the microscopical sections exhibited.

Mr. McADAM ECCLES said that two years ago he had a somewhat similar case. The patient was a woman, aged about 46, who for the previous three years had had symptoms of gastric trouble. When he saw her she was exceedingly thin, and a swelling in the region of the stomach could be easily palpated. Exploration showed a stomach which bore a strong resemblance in its chief characters to that Mr. Moynihan had just shown. It was longer and did not present the marked constriction between the cardiac and pyloric portions, and he found, as in the present case, that there were practically no adhesions, and it seemed a suitable case for gastrectomy. He did not regard his own case as a complete gastrectomy, as a little of the cardiac end of the stomach was left. He proceeded in the same way as Mr. Moynihan had, by ligaturing vessels as far as possible first of all: the coronary artery first, the pyloric artery second, and then dividing the duodenum beyond the pylorus. He then ligatured the right gastro-epiploic artery, and, turning the stomach over to the left side, was able to expose the cardiac portion almost to the œsophagus. Then he considered the difficulty which had been so graphically shown might be overcome by traction, namely, how to manage the suturing of the œsophagus to a portion of the intestine. He, however, boldly cut through the cardiac portion of the stomach just below the œsophagus, and finally ligatured the vessels on that side, and then he was able, the duodenum being exceedingly loose, to bring the duodenum to the œsophagus and do an end-to-end suture between them. He regarded this as a happy chance in this particular case, and it was not to be expected to occur in every instance. The patient did very well and was able to take solid food within three weeks. She left the hospital in a month. She only lived another eleven months, dying apparently of secondary growths in the liver. No post-mortem was allowed, unfortunately, so that he was not able to see the results of the partial gastrectomy. He would not detain the meeting with the microscopical characters, as they were almost exactly the same as those demonstrated in the present case. The point of particular interest in Mr. Moynihan's case was the method of anastomosis between the distal end of the œsophagus and the first loop of the jejunum. It seemed a very practical and useful suggestion in such cases, few and far between, in which gastrectomy could be performed.

Cases of Cholecystectomy.[1]

By John D. Malcolm, F.R.C.S.Edin.

I have removed the gall-bladder for the most part only when it seemed urgently necessary, and on only seven occasions, but all the patients are so well that I think I would have done better if I had performed the operation more frequently.

Case 1.—My first case was that of a woman, aged 56, who was sent to the Samaritan Free Hospital under my care by Dr. Tresilian, of Enfield, in 1902. The gall-bladder was full of stones, and was extensively adherent, its mucous membrane being inflamed and much thickened. When the stones were removed I was unable to pass a probe into the cystic duct, and this fact, together with the unhealthy state of the mucous membrane, induced me to remove the gall-bladder. A large vein close to its base was seen and avoided, and the duct was isolated and tied, two small arteries being also ligatured. A temporary drainage tube was put in, and a good recovery was made. Dr. Tresilian writes that he has been consulted little by the patient since her operation, but she looks very well, and may be considered a cure. After the bladder was removed I was still unable to introduce a probe from it into its duct, but could pass one easily from the duct to the bladder, there being a valve-like projection at the junction between the two. As the valve opened outwards, drainage of the bladder in this case would have led to a free escape of bile, indicating that the ducts were patent; but if the sinus had closed, symptoms of blocking of the cystic duct would have been certain to arise. The complete operation was, therefore, essential to success.

Case 2.—My second case was that of a lady, aged 29, who led a very active life and never had any illness except occasional indigestion pains until 1898, when she noticed a lump in her abdomen. She was never jaundiced. The doctor in attendance, and a surgeon who kindly gave me these details, found the lump so mobile that all attempts to locate its origin were futile. In June, 1899, an exploratory incision showed that it was a very tense gall-bladder, "shaped like a banana."

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[1] Some of the views expressed in this paper were stated in the debate on cholecystectomy at the Exeter meeting of the British Medical Association, and published in the *British Medical Journal*, 1907, ii., p. 877; but the paper was practically complete before that meeting, and the Council of this Section has kindly allowed me to republish these views with the cases.

The fluid and some stones were removed through an incision which was sewn up. The bladder was returned to the peritoneal cavity, and the latter was closed without drainage. Except a slight swelling of the left leg, there was no trouble during convalescence, and the patient left the nursing home at the end of three weeks. Afterwards she suffered much from indigestion, occasionally with fever, and I was consulted on March 8, 1902, on account of a severe prolonged attack of pain and vomiting. The gall-bladder was then enlarged, hard, tender, and fixed to the back of a short median incision half-way between the umbilicus and the xiphoid cartilage. The temperature was steadily above 100° F. Rest and soothing treatment eased the pain without giving real relief. After consultation with Sir Douglas Powell, I reopened the abdomen on March 15, 1902. The gall-bladder was distended by white fluid—its own secretion—which was removed with some half-a-dozen stones. It was fixed in a bent position by adhesions, so that its lumen was narrowed, and I could not pass my finger into its deeper parts without considerable separation of adhesions. I had told the patient that I might drain the bladder and remove it later, and I decided to adopt this course. The temperature was normal two days after the operation, and convalescence was uneventful. Some more stones escaped, but no bile at any time. The opening was maintained by a drainage-tube. I did not anticipate a long interval between the operations, but the patient was out of town a great deal until November, 1902, when I removed the gall-bladder. There was a calculus in the cystic duct, and it was necessary to divide the latter close to the common duct. Convalescence from this third operation was complicated by a return of the swelling of the left leg which developed after the first operation, and there was some weakness of that limb for a time, but I saw the patient recently and she was then very well. In this case the obstructing calculus was fixed between two of the folds of mucous membrane which form an imperfect spiral ridge within the cystic duct, and which were unusually well developed. The stone appeared to be sufficiently loose to permit of the passage of fluid under pressure, so that the contents of the bladder had slowly escaped, and the condition of prolonged tension and tenderness without any more serious development, when I first saw the patient before her second operation, was fully explained.

Case 3.—The third case was that of a man, aged 56, whom I was asked to see by Dr. R. D. Mackintosh, of Mortlake. He was of very active habits and enjoyed all kinds of outdoor exercise, but had suffered from indigestion for many years. In December, 1903, he had an attack of unusually severe pain, with fever, and Dr. Mackintosh diagnosed gallstones. On the 29th of that month I removed a stone from an intensely inflamed adherent gall-bladder. The stone measured 1¾ in. by 1¼ in., and was shaped like a barrel with one end pointed. I decided to drain for a time, and the bladder was removed on February 4, 1904. The cystic duct was surrounded by dense adhesions, and was distended almost to its junction with the common duct, so that it had to be ligatured close to the latter. The patient made a perfect recovery and has continued well. Since these notes were handed to the Secretaries Dr. Mackintosh informs me that this patient, on November 2, was seized with severe pain over the gall-bladder region, accompanied by vomiting of bilious matter. This continued for eight hours, and was followed by jaundice, high-coloured urine, and putty-coloured stools, which persisted for four days. No stone was detected. There has been no recurrence of pain or sickness. At the time of this attack the patient was suffering from an acute nasal catarrh, and Dr. Mackintosh suggests that the illness may have been due to a catarrhal jaundice. He adds that "the general condition is splendid, the operation having cured him of persistent dyspepsia, 'sciatica,' and vague lumbar pains." The patient is a very athletic man, whose occupation necessitates daily prolonged office work.

Case 4.—My fourth case was that of a woman, aged 43, who was sent to me at the Samaritan Free Hospital by Dr. C. H. J. Watson, of Reigate. The history and conditions were very similar to those of Case 3. There were twenty-two stones, all measuring about ½ in. in their longest axes, of an ivory-white colour, and dotted over with numerous round brown spots about two lines in diameter. One was fixed in the cystic duct. The treatment was similar to that in Case 3, and there was the same necessity for tying the cystic duct very close to the common duct. I intended to remove the bladder at the first operation in this case, but on beginning to separate it the hæmorrhage was so free that I decided to drain first. In November of this year the patient described herself as "quite well."

Case 5.—I saw the next case in consultation with Mr. J. Kingston Barton. The patient, whose age was 47, had suffered from attacks of sickness, with vomiting, for quite twenty years. She had a swelling over the appendix region which appeared to be cystic and somewhat mobile. There was an area of tenderness behind this swelling and apparently separable from it. After a careful examination we came to the conclusion that the gall-bladder was enlarged and displaced downwards, and that the appendix was inflamed. Both were removed, each through its proper incision, on November 10, 1904. The gall-bladder was of oval shape, about 4 in. in length, with very thin walls, and quite free from any adhesions. It contained two stones, but its distension was due to an elongation and narrowing, almost to obliteration, of the cystic duct. Probably the condition was congenital and had become aggravated by the traction of the gradually enlarging bladder. The patient made a good convalescence. The attack of appendicitis was a fortunate development in this case, as it led to the removal of the gall-bladder before the changes in it gave rise to dangerous symptoms. An inflammatory attack in such a thin-walled bladder, lying amongst the small intestines, might have given rise to very serious complications. Mr. Barton tells me that this patient's health was completely restored six months after the operation, that she has remained well ever since, and that there have been no more attacks of sickness.

Case 6.—The sixth case was sent to me by Dr. J. F. L. Whittingdale, of Sherborne, in 1905. I propose to publish it at greater length elsewhere. Shortly, the gall-bladder was universally adherent and a fistulous communication had formed between it and the stomach, whilst the common and hepatic ducts were dilated and contained stones. The gap in the stomach-wall was closed and the gall-bladder was drained on January 24, 1905. The gall-bladder and the stones in the common and hepatic ducts were removed on February 28, 1905. The patient made a good recovery, and remains well.

Case 7.—The last of these cases was that of a lady, aged 52, who had jaundice at the age of 5 years, and suffered much from pain and indigestion until she was 13. When her age was 17 years she had severe abdominal pain, with jaundice and fever, continuing for ten weeks. Gall-stones were diagnosed, and the patient's father, who was a medical man, decided that she should be operated upon, but the pain abated and nothing was done. After recovering from that illness the patient, who was of a nervous temperament, had no symptoms that were definitely attributed to gall-stones. In 1899 an attack of appendicitis developed, and I was present when the inflamed appendix was removed. The gall-bladder was found to contain thirty-six stones, from $\frac{1}{4}$ to $\frac{1}{2}$ in. in diameter, which were removed through a second incision. The opening in the gall-bladder was sewn over with interrupted silk sutures, and it was dropped into the peritoneal cavity, which was closed without drainage. Union was immediate and recovery was good, although there were some temporary nervous manifestations, including an unexplained pain near the cicatrix, which only very gradually ceased. Five years later the patient again complained of discomfort rather than pain in the gall-bladder region, with tenderness on palpation and a slight rise of evening temperature. These evidences of mischief were persistent, although prolonged complete rest was taken. It was therefore decided to remove the gall-bladder, and this was done in the spring of 1905. There were slight adhesions, but the cystic duct was normal externally. The operation offered no difficulties, the patient made a good recovery, and she remains well. The gall-bladder showed a scar ½ in. long on its mucous surface. There were three silk ligatures within it, all of which were coated with a thin layer of calculous matter. They had been used to close the opening in the gall-bladder at the first operation and had been discharged into it. Close to the cystic duct there was a small piece of tissue, resembling granulation tissue, attached to the mucous membrane, and which had probably caused a partial valve-like obstruction. The specimen is preserved in the Museum of the Royal College of Surgeons (No. 2830 P., Pathological Series).

A consideration of these cases leads to the conclusion that the excision of the gall-bladder is an exceedingly satisfactory operation. One of the chief advantages of removing it, as compared with its evacuation and temporary drainage, might seem to be the impossibility of a further formation of gall-stones in it; but the probability of a new development of stones in this viscus after it has been cleared of them is not great. I have in my possession a letter in which the late Mr. Knowsley Thornton wrote that he had never known gall-stones to form again in the bladder after cholecystotomy. I was surprised by this assertion, but I could not produce any evidence that the experience was unusual, and in agreement with it Mr. Mayo Robson has recently stated that in his practice, and in that "of several surgeons whose aggregate of operations on the gall-bladder amount to fully 3,000, it is universally acknowledged that the recurrence of gall-stones after cholecystotomy is an extremely rare event."[1]

I have removed stones from the gall-bladder after it was supposed to have been cleared, but I pointed out, in 1895, that it may be difficult to be sure that stones have not been overlooked at the first operation.[2] I have seen only three cases in which there seemed to be little doubt that calculous matter did form in the gall-bladder after a cholecystotomy. In one instance, No. 7, related above, the pieces of silk in the bladder made the circumstances so peculiar that the case does not contradict the rule that recurrence is rare.

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[1] *Brit. Med. Journ.*, 1906, i., p. 430.

[2] *Trans. Med. Soc. Lond.*, 1895, xviii., p. 248.

In another case Mr. Knowsley Thornton removed a number of stones from the gall-bladder of a lady, aged 32, and with the exception of one attack in 1890 she was free from definite symptoms of gall-stones for ten years. In October, 1899, she was again seized with biliary colic, and I removed a considerable quantity of calculous matter of the consistence of partially dried putty. The stones had firm shells, but the slightest manipulation broke them in pieces, so that none were removed whole. The bladder was drained, and as the ducts became clear the wound was allowed to close, and there has been no recurrence of symptoms that could be definitely attributed to gall-stones. I had an opportunity in 1904 of examining the parts in performing a hysterectomy on this patient, and found no stones. There is, of course, no proof that calculous matter was not left behind at the first operation in this case. The third case of re-formation of gall-stones is related below.

It would seem, therefore, that the risk of a new development of these stones is not a strong argument in favour of removing the gall-bladder. But in my first case it was quite clear that a simple drainage would have been followed by symptoms of obstruction in the cystic duct, indistinguishable from those due to an impacted stone, and the following case is an example of conditions in which I would have acted more wisely in the interests of my patient if I had removed the gall-bladder. A woman, aged 49, had suffered from attacks of abdominal pain, frequently accompanied by jaundice, for twenty-two years, being dangerously ill on at least two occasions. In 1901 she consulted Dr. Herbert C. Male, who brought her to me. On exposing the gall-bladder when the patient was comparatively free from pain, it was found to contain many calculi, but it was not distended. I incised the fundus, and a cavity was disclosed which was occupied and filled by a single stone, measuring about ½ in. in its longest diameter. I opened the main cavity of the bladder by cutting away the smaller one with the septum between the two. The deeper part was then cleared of stones and drained in the usual way. The sinus was allowed to close, and the patient made a good recovery and was free from pain about a year, but since then she has at times suffered from severe biliary colic and has passed several stones, some quite of large size. The long history and the evident tendency to cicatricial contraction of the bladder-wall suggest that its removal would have given a better result. This operation should, I think, now be done, but the patient refuses to consider another operation. There was no obvious communication between the cavity containing the single stone and the rest of the gall-bladder, but Mr. W. S. Handley, who assisted me at the operation, succeeded in getting the section drawn in fig. 1, showing that a sinus remained between the two. Fig. 2 is a sketch of the part removed. It was drawn by Mr. J. P. Stephens Ward, of Plymouth, who was interested in and present at the operation. Fig. 3 shows diagrammatically the hour-glass constriction which existed.

Since I put these notes together it has been argued by Mr. Bland-Sutton that, in order to avoid fresh trouble, it is desirable to remove the gall-bladder whenever it is interfered with surgically.[1] One objection to this course is that the removal is more dangerous than the drainage. This, however, is not such a strong argument as it might seem. The excision of a fairly healthy gall-bladder is, in such cases as my fifth and seventh for example, an extremely easy and safe procedure. The increased risk is not appreciable unless the gall-bladder is much disorganised by inflammation. But it is in cases of prolonged inflammation that a simple drainage most often fails to effect a cure, and thus, when the danger of the radical operation is greater, this procedure is

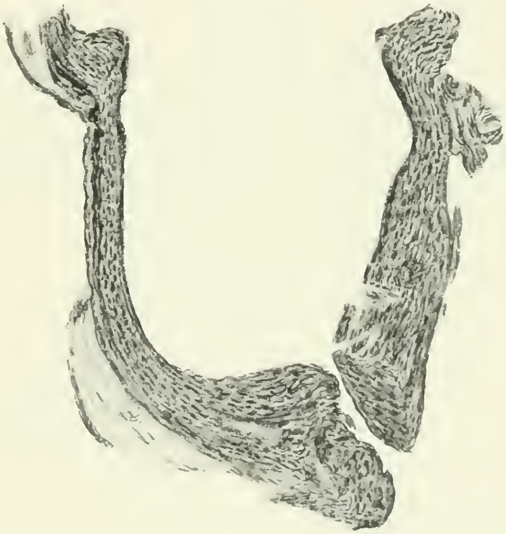

Fig. 1.

Section of part removed, showing communication between the outer cavity and the main cavity of the gall-bladder.

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[1] *Brit. Med. Journ.*, 1907, ii., p. 877 (Report of Exeter meeting of the Brit. Med. Association).

proportionately more necessary. The increased danger of its removal is therefore not always a good argument for preserving the gall-bladder.

A better reason is that its absence does away with a convenient and safe means of draining the gall-ducts either on to the skin or into some portion of the intestine. This may become a very important consideration if an obstruction of the common duct should arise, and, although it is only occasionally that the bladder would be absolutely essential as a substitute for the common duct, the possibility of its being required for this purpose seems sufficiently important to make it undesirable to sacrifice the healthy gall-bladder simply because stones have formed in it, especially as a redevelopment of calculi is rare, and I think particularly rare in a healthy gall-bladder.

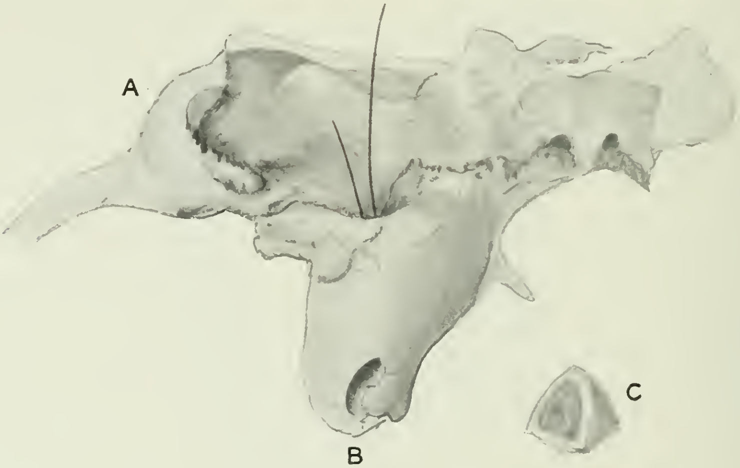

Fig. 2.

Sketch of part removed. **A** is immediately above a strong adhesion to the anterior abdominal wall. The bristles enter the widely opened cavity in which the stone, **C**, lay. **B** is at the line of section of the gall-bladder where the walls have fallen together.

In several of my cases I have drained the gall-bladder and removed it at a later date. This plan, for obvious reasons, should be restricted in its application as much as possible. But it is a well-established fact that in operating on the vermiform appendix the danger is greater if surgical interference is necessary when an acute inflammation of that part exists, and the same statement applies to operations on the gall-bladder. Moreover, in the case of the gall-ducts, the surgeon is very frequently able to give instant relief from all pain and inflammatory mischief by draining the bladder. This treatment, as a rule, is hardly accompanied by any danger, and at a subsequent date calculi in the larger ducts or the gall-bladder itself may be removed under the safest conditions.

A serious risk from interfering with an inflamed gall-bladder was strongly impressed upon me by a case of fatal hæmorrhage which I had the misfortune to meet with twelve years ago. An elderly lady who had suffered from gall-stones for many years was suddenly seized with intense pain in 1895, and rapidly became deeply jaundiced. She did

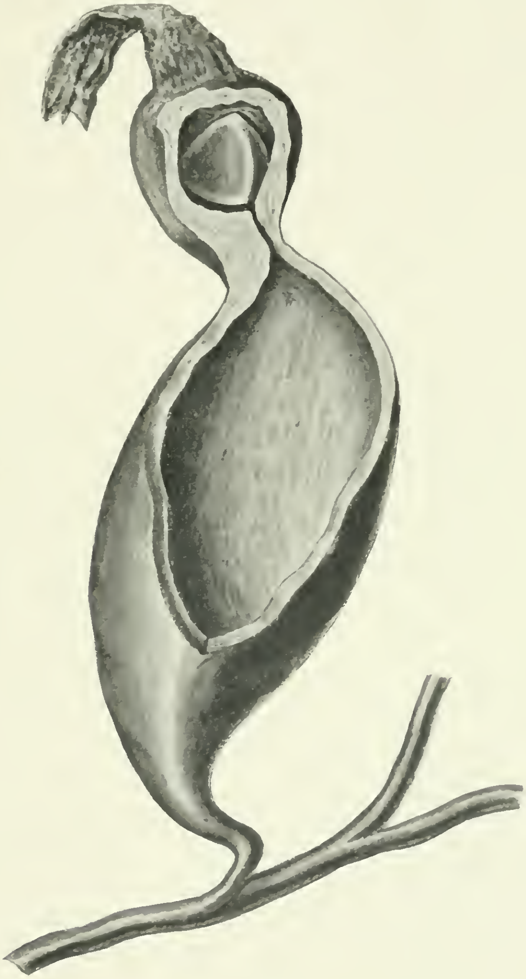

Fig. 3.

Diagrammatic representation of the parts showing the hour-glass constrictions and a strong adhesion to the fundus.

not, as on other occasions, get relief, and after twelve days she had a rigor, with a rise of temperature to 106° F. She was then seen, in consultation with her medical attendant, by a physician, who recommended an immediate operation, but further consultations were desired, and I did not see the patient until there had been rigors, followed by a temperature of 106° F. or higher, on four consecutive evenings, the morning temperature being normal or subnormal. The gall-bladder, when exposed, had all the appearance of sloughed tissue. Adhesions were universal, but recent and easily separated. The bladder was distended by greenish fluid, and when it was emptied a stone was detected in the common duct, with a projection blocking the cystic duct, which was dilated so as to allow my finger to touch the stone. The latter, which appeared to be about $\frac{3}{8}$ in. in diameter, was easily felt with the fingers in the peritoneal cavity. As no bile was coming into the gall-bladder, and none had passed into the intestine since the illness began, it seemed desirable to remove this stone. It was therefore broken up with the point of a knife, introduced through the cystic duct, and taken away piecemeal. In manipulating the parts, with a view to pushing forward the stone, my fingers sank into the sloughy tissue covering it, but not so far as to touch the stone, and from the bruise thus made an uncontrollable hæmorrhage took place. It did not arise at once, but with the removal of pressure from within the gall-bladder that viscus regained its vascularity, and as I sutured it to the abdominal wall every needle puncture bled freely. It was whilst I was securing the bladder in position for drainage that the serious hæmorrhage was noticed. I divided the right rectus muscle and got a good view of the parts. There was no bleeding point, but only a general oozing from the damaged tissue. It was impossible to place a ligature round the affected area without including the hepatic and common ducts, and the hæmorrhage was too profuse to permit of the action of styptics. Pressure was ineffectual, and when I attempted to pass a ligature through the tissues every needle puncture caused more bleeding. Dr. Andrew Elliot, who was assisting me, aided by Dr. E. M. Callender, who gave the anæsthetic, very rapidly effected the infusion of saline fluid into a vein, but of course this was useless when the hæmorrhage was not arrested.

Uncontrollable hæmorrhage is described as one of the dangers of operations on jaundiced patients, and it has been attributed to a change in the blood caused by the presence of bile in it, in consequence of which coagulation is less rapid and firm. I have long observed that in jaundiced patients an incision through the abdominal wall is generally not accompanied by free or peristent hæmorrhage or other evidence of want of coagulability of the blood, and in the case under consideration the abdominal wall was particularly bloodless. This is the only case of fatal hæmorrhage that I have seen in any operation on the gall-ducts, and the conditions seem to confirm the view that its cause is local rather than general. John Hunter asserted that bile, when mixed with the blood, prevents coagulation. But he added that "we cannot suppose that in a living body it can be taken into the blood in such quantity as to produce this effect; for we find in a very severe jaundice that the blood is still capable of coagulating strongly."[1]

In the case I have recorded, and in a minor degree in all cases of distended and inflamed gall-bladder, there are good reasons why hæmorrhage should be a danger apart from the condition of jaundice. A focus of inflammation is always surrounded by an area of enlarged blood-vessels, and the engorged liver must offer an obstruction to the blood-flow. In the case related above there was practically no circulation through the gall-bladder because of the inflammation and pressure within it. When the tension was removed the blood re-entered the wall of the gall-bladder, and the blood-vessels in it seemed to have temporarily lost their tone and so permitted an uncontrollable loss of blood from the bruised tissue at the point of juncture between the sloughing and the healthy but congested structures.

All authorities are agreed that shock leads to a distension of the large veins of the body, a condition which must also conduce to a free hæmorrhage from torn tissue in the central area of the portal system.

Hence, whilst every effort should be made to increase the coagulability of the blood before operating on a case of jaundice, a most important point in the surgery of the bile-ducts is to avoid cutting, and especially tearing or bruising, of tissues from which hæmorrhage cannot be completely controlled by ligatures, and the urgency of this is much greater when the tissues are inflamed. I attribute the hæmorrhage in the case I have recorded more to the condition of the tissues than to the condition of the blood. In this connection the necessity for seeing the parts operated upon, so strongly urged by Mr. Mayo Robson, is also important.

It seems to follow that in cases of acutely distended and inflamed gall-bladder it must be safer to be content to drain the bladder until inflammatory conditions subside, unless it is possible to pass a ligature completely beyond the affected area. In the case I have described I thought that the blocking of the cystic and common ducts necessitated the removal of the stone, but further experience has led me to believe that the relief of tension from draining the bladder would have been followed by an amelioration of symptoms and an escape of bile. The rapid return of blood to the apparently dead bladder-wall showed that there would have been no danger from gangrene. Of course, if the stone had been in the cystic duct and not too close to the common duct, a ligature could easily have been applied so as to control the hæmorrhage.

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[1] John Hunter's Works, edited by J. F. Palmer, 1837, vol. iii., p. 35.

Another point of practical interest in considering these cases is the frequency with which the cystic duct is dilated to, or very nearly to, its junction with the common duct, so that the surgeon must not count upon finding the inch and a half of narrow cystic duct described in anatomical works in which to choose his place for ligaturing and dividing it.

For the reasons which I have given, it seems to me that the removal of the gall-bladder should be undertaken in most of the cases in which calculi have been left so long in the body that the bladder is much disorganised. There is a strong temptation to remove it in early cases also because of the ease and safety with which this may be done and the greater rapidity of convalescence, but this procedure seems to be unnecessary in such cases, and it greatly increases the danger if drainage of the remaining ducts should be required on another occasion. Apart from the occasional extreme difficulty of the operation, this possible need for the gall-bladder to act as a drain is the only real objection to its removal in every case in which it is interfered with at all, and the ultimate decision on this question must always rest upon the estimated danger incurred by the want of the bladder as a means of drainage, as compared with the risks of fresh mischief arising from its presence and from a second development of stones in it. Whatever the final decision may be, the greatest necessity in connection with these cases at present is that operations should be performed earlier. In one of my cases (Case No. 7) the history points to the existence of calculi during more than forty years, and a record of symptoms lasting over twenty years is not uncommon. If the conditions were diagnosed promptly and the patients would submit to operative treatment between the attacks, as in cases of appendicitis, the most urgent reasons for removing the gall-bladder would, to a very great extent, be obviated. The comparatively safe operation of evacuating and draining a healthy gall-bladder would be frequently substituted for the much more dangerous treatment of the inflamed parts, and would be practically without a death-rate, whilst those who think it right to remove the gall-bladder in all or most of these cases could do so with a very small risk.

Mr. MOYNIHAN said his difficulty always was, when asked to state his reasons for removing or leaving the gall-bladder, that he found there were so many factors to take into account that it was very difficult to put his practice into a few words. He had now removed the gall-bladder about 110 times, but his feeling was that, although he now removed a gall-bladder oftener than he used to, he did so relatively less frequently. The phase of mind he went through on the subject was that the more experience he had the more conservative he became in regard to the removal of the gall-bladder, and that unless there were definite indications to the contrary, it should be left. For example, if there were malignant disease of that viscus, or chronic cholecystitis to a degree which made the mimicry of malignant disease so striking that it was impossible to say without seeing the specimen under the microscope that the condition was not malignant, or if there were fistulæ between the gall-bladder and the colon, or between it and the duodenum, where the cystic duct was involved, either by a stone which had become impacted there a long time or by stricture resulting from a stone which had passed, removal of the gall-bladder should be done straight away, because unless it were done certain conditions would be left which would have to be dealt with in the future by a further operation. But there were an increasing number of cases in which it was certainly not right to remove the gall-bladder. It was important—and Mr. Malcolm had not mentioned that point—to take into consideration the physical condition of the patient. It was not a question of whether it was better to do a cholecystectomy or whether it was better to do a cholecystotomy, because of the conditions in the gall-bladder, but what it was right to do for the particular patient. One might have to remove the gall-bladder in a case in which one felt that cholecystotomy would be the safer operation, though it would not relieve the patient to the same extent. But there were also cases, especially in very stout women, the subjects of chronic bronchitis or asthma, in which one was compelled to leave the gall-bladder because, although not such a satisfactory operation as cholecystectomy would have been in that case, the risk of the more mechanical details of the operation, the dragging and elevation of the liver and the lengthening of the incision, would make the operation of cholecystectomy a much more serious one. That was a point which was often ignored in discussions on the subject. Anybody who had been face to face with an excessively stout patient, with perhaps a fatty heart and chronic lung conditions, and who also had perils from trouble in the gall-bladder, would know that although one felt that removal of the gall-bladder was the thing to do so far as the mere disease was concerned, it would really be nothing short of criminal to do it in that patient. There was a good healthy English prejudice in favour of not doing more than was indicated in a particular case. It was very satisfying to speak of the removal of the gall-bladder preventing the possible recurrence of stones. He had great sympathy with that view, because he expressed it himself years ago in a paper on cholecystectomy, in which he advocated the almost routine performance of the operation. But he had now come to the feeling that unless the indications for cholecystectomy were definite and positive, it was right and proper to perform cholecystotomy in preference. He regarded the recurrence of gall-stones as excessively rare; he much doubted whether there were half a dozen genuine cases of it in all the literature. The leaving behind of gall-stones was not an infrequent procedure. He wished to say that, with the exception of some complete gastrectomies, there were particular difficulties in regard to removing gall-stones which were not approached in the surgery of any other part of the body. On the previous day he was operating, in the presence of a distinguished surgeon, upon a patient who had gall-stone troubles. He gave the recent history of the case, and the surgeon said he supposed the operation would be cholecystotomy. It was not a fair question to ask, because he did not believe in asking a man what was the right thing to do. But the history was so clearly that of ordinary recurrent attacks of gall-stones that he said the patient had a stone in the common bile-duct. It turned out that he had to do a cholecystectomy, partial hepatectomy and choledochotomy, the double operation, opening the duodenum to remove a stone from the ampulla, in a patient who apparently had nothing more than a stone which required cholecystotomy. The patient had malignant disease of the gall-bladder in the early stage, a stone embedded in the liver, a stone in the common duct, which was very much dilated, and also a stone in the ampulla which he could not dislodge. He quoted that case merely to illustrate the excessive difficulties there were in cases of that type, and how impossible it was before commencing the operation to state exactly what would be the nature of the operation. Some of the most expert surgeons had overlooked conditions which they did not expect to find, and it need hardly be said that removal of the gall-bladder added an element of the greatest possible significance to any future operative procedures which might have to be carried out. If the surgeon could be sure in all cases that there were no stones in the duct or in the liver high up, the removal of the gall-bladder might be carried out more frequently than at present; but the difficulty seemed to be to make quite sure that, in removing the gall-bladder, even when the case was a proper one for that operation, there was not something left behind. That was why, when beginning that work, a surgeon should not be persuaded to do cholecystectomy unless there were very grave and advanced disease of the gall-bladder; he should be content with cholecystotomy until he had become quite familiar with that particular region of the body. Even then, even with the experience of a Malins or a Mayo Robson, it was excessively difficult to be sure that there might not be some condition remaining behind which would have made cholecystotomy preferable to cholecystectomy. When surgeons, who were watching him operate, asked him his views on the subject now under discussion, he was accustomed to reply that it would be necessary for them to go to his house to talk the matter over.

Surgical Section.

February 11, 1908.

Mr. J. Warrington Haward, President of the Section, in the Chair.

Coxa Valga (Collum Valgum).

By A. H. Tubby, M.S.

While coxa vara has received much attention during the past few years, very little has been directed to the opposite condition, namely, coxa valga, and notices of it are sparsely scattered throughout surgical literature.

Starting from the conventionally accepted conception of a varus deformity as one in which the segment of the limb distal to the curve is found adducted, we call that form of bending of the neck of the femur coxa vara in which the head is displaced downwards and the shaft of the femur adducted. Coxa valga is exactly the opposite condition to coxa vara. The head of the femur is displaced upwards and the shaft is abducted.

The cardinal signs of coxa valga are abduction of the leg associated with external rotation and limitation of adduction. These, taken with other symptoms and signs to be detailed presently, make up the "symptom-complex."

In effect coxa valga is an opening out of the angle made by the head and neck of the femur with the shaft, but it has not received much notice hitherto owing to the fact of its sometimes being mistaken for early hip-joint disease; and the clinical symptoms to which it gives rise have been hitherto considered to be of slight importance.

Before we proceed to the description of the affection we must first render clear the terminology. Most of this has been gradually evolved in the descriptions of coxa vara, but the same terms and measurements are applicable to and in use in descriptions of coxa valga.

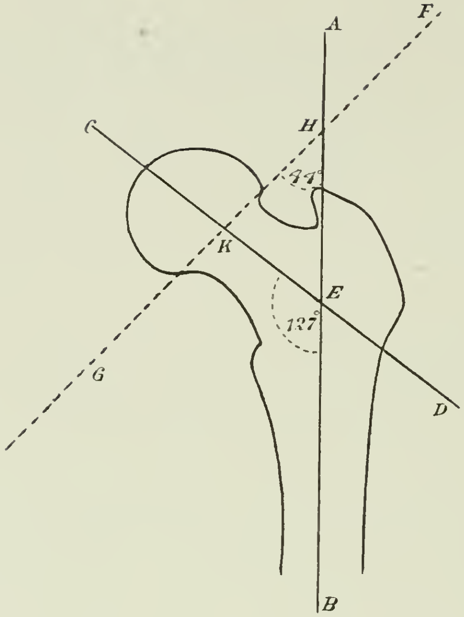

FIG. 1.

NORMAL FEMUR.—Angle *C E B* = angle of inclination or angle of depression; *K H E* = Alsberg's angle of elevation; triangle *K H E* = Alsberg's triangle.

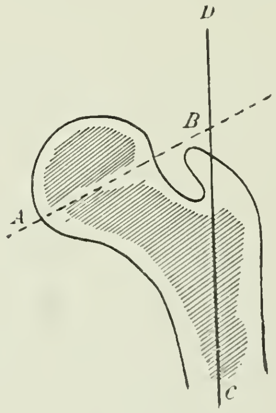

FIG. 2.

A B = Hoffa's line; *C D* = axis of shaft of femur; angle *A B C* is Alsberg's angle, or angle of elevation or angle of direction.

If we examine the upper part of the normal femur and make our measurements in a frontal plane, drawing one line (*A B*) through the long axis of the shaft and another line (*C D*) through the long axis of the head and neck, we have an angle (*C E B*). This is called (1) the angle of inclination, or the angle of depression, the cervical angle, or angle of the femur. It varies from 125° to 128° in the normal. Anything above 128° represents coxa valga, and anything below 125° represents coxa vara (fig. 1). Again, taking the normal femur, and drawing a line (*A B*, fig. 2) through the base of the head at a point where the cartilaginous covering ceases, we have (2) Hoffa's line. This line (*A B*) is represented in fig. 1 by *G F*, and cuts *C D* at *K*. (3) Alsberg's triangle. The triangle made by the lines *K H*, *H E* and *E K* is Alsberg's triangle (*see* fig. 1). (4) Alsberg's angle, or the angle of direction, or the angle of elevation, is the angle formed by Hoffa's line, drawn through the base of the head of the femur, and the line continued up from the diaphysis, indicated by the lines *K H* and *H E* in fig. 1, and by the lines *A B* and *B C* in fig. 2. It is normally about 41° to 44°.

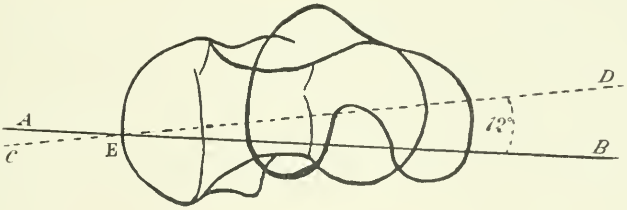

Fig. 3.

Angle of Declination.—Femur standing on table, looked at from above down. *A B* = transverse axis of knee-joint (or line parallel thereto); *C D* = long axis of neck; *D E B* = angle of declination = about 12° normally.

Inasmuch as in coxa vara there are two elements constantly present, namely, depression of the head and neck of the femur, another term has come into use, namely, the angle of declination (fig. 3), which is measured in the following way: The femur is stood vertically on the table, and looked at from above downwards. A line (*A B*) is drawn through the transverse axis of the knee-joint, and a line (*C D*) is drawn through the long axis of the head and neck. The angle *D E B* is the angle of declination, and is normally 12°. It is increased in some forms of coxa vara and in congenital dislocation. There is reason to suspect that when we know more fully the pathology of coxa valga we shall find not only that there is an elevation of the head, but that there is also a distinct curvature forwards or backwards of the neck, and even, as is described in coxa vara, a torsion of it upon itself. Probably the latter condition will be found to preponderate over curvature. It is simpler to deal with one measurement in discussing coxa valga, the angle of inclination, or the angle of depression,[1] or, for brevity, the angle of the femur. In some cases of coxa valga the angle may be increased so much that in X-ray pictures, with the limb everted, the neck of the femur may appear to be in a continuous line with the shaft (*see* cases I. and II., figs. 10 and 11).

MUSEUM SPECIMENS OF COXA VALGA AND CASES DESCRIBED IN LITERATURE.

Coxa valga was first recognised in museum specimens. The late Sir George Humphry, in 1888 [12], speaks of four specimens in the museum of the University of Cambridge which he examined. The first specimen is seen in the skeleton of a child with hydrocephalus, the angle of inclination of the neck of each thigh bone being 148°, the length of the bones being 9¼ in. The second is the upper part of a thigh bone, which was taken from a limb removed at the hip-joint. The patient had been paralysed from infancy. In the third specimen the angle of the neck was 150°, and it was judged to have been taken from a paralytic limb. The bone measured 16 in. in length. The last was an amputation stump, in which the angle of the neck was 142°, and it was inferred that the limb had borne no weight for some time previous to the operation.

Albert [1] mentions eight specimens: one associated with paralysis of the legs; one with "weakness" of the legs, but no paralysis; one with osteomyelitis of the pelvis; one with rickets; one with osteomalacia; one with multiple exostoses; one with luxation (congenital ?) of the other hip; and one with genu valgum.

Turner [22] mentions three specimens: one from an amputation in early life, with an angle of 150°; one associated with tuberculosis of the ankle and knee; and one from fracture of the lower end of the femur, with separation of the epiphyses.

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[1] On the whole the term "angle of inclination" is preferable in discussing coxa valga. The expression "angle of depression" is of value in speaking of coxa vara, because the neck and head of the femur are depressed, whereas in coxa valga the contrary is the case, and, therefore, "angle of depression" is apt to be misleading.

Lauenstein [14] notes three specimens, with angles of 140°, 146° and 155°; and he observed increase in the angle of inclination after amputation through the thigh.

The cases found in literature are the following:—

Mauclaire [17], to whom we owe the term "coxa valga," described the condition as occurring in scoliotics, especially those with a right dorsal convexity. In March, 1894, he performed an autopsy of such a case, and the neck of the left femur showed an angle of declination of 110°, or an angle of inclination of 175°. During life internal rotation of the limb was impossible and adduction was greatly limited.

Hofmeister [11] observed the condition in a rickety case.

Thiem [19] found coxa valga following injury to the epiphysis of the neck in a child.

Hoffa [9] records a case of coxa valga after fracture of the neck of the femur.

Reichardt [18] mentions one case occurring in connection with infantile paralysis. The case was a girl, aged 13, who had bilateral club-foot since birth and acute anterior poliomyelitis. The right trochanter was 5 cm. and the left 4 cm. below Nélaton's line.

Lauenstein, Gangolphe and Hau [15] refer to a man, aged 20, who had septic disease in the right knee, causing genu valgum on the same side, and subsequently septic disease in the left hip, followed by coxa valga in that hip.

Hoffa [9] refers to a case following infantile paralysis.

David [5] speaks of a boy, aged 5, who early in 1903 was treated for pseudo-hypertrophic muscular palsy; both limbs were markedly abducted, and the trochanters were in Nélaton's line, while adduction and inward rotation were diminished.

Young [23] describes five cases of his own. The first occurred in a boy, aged 8, after traumatism; and Young surmises that fracture of the neck of the femur occurred five years previously, when the child fell and the mother noticed he "walked crooked." The details given of the case are as follow: The patient stands on the left leg, with the right knee thrown forward and inward, the lumbar spine curved, with the convexity to the right, and the left shoulder depressed. In walking he limps on the right leg. In standing the creases of the buttocks are inclined to the left, and the abdomen is pendulous and prominent. In the lying position there is slight lordosis, and the pelvis is tilted upward on the right side. The right leg is apparently longer than the left, although the bony measurements are the same. On flexion of the right hip the thigh is carried outwards, and adduction is then limited. In the left hip all the movements are normal. The right leg is adducted upon the pelvis. Flexion of both limbs shows the femur of the right leg to be a little longer than that of the left, and the lordosis is not entirely lost until both thighs are flexed upon the abdomen. Measurements taken from the skiagraphs show the angle of inclination (depression) on the right side to be 142°, while on the left it is 132°. Young adds: "I divided the adductors from their pelvic attachments, encased the limb in plaster, and abducted the limb for two weeks."

With regard to this case there is one detail which appears to be entirely contradictory. The statement that the "right leg is adducted upon the pelvis" is at variance with the usual experience in these cases. A limb affected with coxa valga is abducted, and not adducted.

"Coxa valga from forceps delivery.—P. R., a girl, aged 4. At birth forceps were applied to the breech until traction could be made on the feet. She did not walk until she was 4 years of age. The condition then was that both limbs were stiff. The feet were held in the equinus position and the limbs were poorly developed. The trochanters were posterior to, and 1 cm. below, Nélaton's line. The limbs were held in the adducted [1] position, and could be only slightly separated. Abduction was very limited, with noticeable spasm of adductors. Internal rotation was possible on the left side, but very slightly so on the right. Flexion of the thighs on the pelvis was difficult. The outline of the thighs and hips was abnormal on both sides. The skiagraph shows coxa valga on both sides, more marked on the right, and backward displacement of the trochanter, also on the right side. On the right side the angle of inclination (depression) was said to be 174°, and on the left side 164°."

"Coxa valga from separation of the epiphysis.—A. R., a boy, aged 4. He fell two years previously and walked about with a limp for one week. He was then examined, and the right leg was found to be shortened 1 in. An X-ray photograph showed a separation of the head and epiphysis of the femur on the right side. The limb was put up in an abducted position and a weight of 5 lb. applied. He was discharged four weeks from the time of injury. On May 6, 1906, he was readmitted on account of stiffness in the right leg, which was more prominent in the morning on rising. He also limped slightly in walking. In a skiagram the angle of inclination (depression) on the right side was seen to be 144° and on the left side 133°."

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[1] As a rule in bilateral cases the limbs are held in the abducted position.—A. H. T.

"Coxa valga, from knee-joint disease.—W. R., a boy, aged 2½, sustained a slight traumatism of the left knee and hip in February, 1903. Extension was applied for three months, and then plaster of Paris to the knee-joint. In October, 1903, an abscess formed, and a superficial sequestrum was removed from the outer condyle of the femur, which contained tubercle bacilli. In January, 1904, the biceps tendon began to contract, and it was for this he sought advice. Examination showed ankylosis of the left knee-joint from tuberculous arthritis, complicated by the clinical symptoms of coxa valga.[1] The affected limb was 1½ in. longer. Skiagraphically coxa valga appeared to exist. On the left side the angle of inclination (depression) was said to be 160°, and on the right side 130°." Young adds: "I divided the biceps tendon and equalised the limbs by a sole on the sound side. The leg became ¼ in. shorter." In what way this occurred is not even mentioned or discussed.

"Coxa valga from rickets.—A boy, aged 4, was seen on April 13, 1906. The mother noticed a peculiarity in the child's walk two months previously, the gait being shuffling, and the right leg being more affected than the left. *On examination:* Nothing important is to be noted in the general condition, save that the epiphyses are slightly enlarged and the abdomen prominent, but there are no other evidences of rickets.[2] No history of injury. The patient stands resting the weight on the left leg, with the right foot slightly advanced and the knee flexed. He walks with a decided limp on the right side. In the prone position the right foot is slightly everted, the knee slightly bent, and the thigh slightly abducted. Forced flexion is limited, abduction extreme, and adduction slightly limited. There is no contraction of the tendons about the knee or hip. The right leg is apparently longer than the left. A skiagram shows the neck of the femur in the right leg to be in a position of coxa valga. On the right side the angle of inclination (depression) is 150°, and on the left side 140°."

Unhappily the descriptions of some of these cases of Dr. J. K. Young's are lacking in essential details, more particularly so far as the clinical signs are concerned, and for his diagnosis he has, in the writer's opinion, too largely relied upon X-rays. The fallacies in connection with this point will be alluded to later.

Relative Frequency.—In order to determine the normal angle of the neck of the femur as well as the relative frequency of coxa valga and coxa vara, measurements were taken by J. K. Young of a large number of specimens in the Museum of the College of Physicians and the Wistar Museum in Philadelphia. These were mostly adult specimens of all nationalities and both sexes. Measurements were made of the angle of inclination (depression) and the angle of declination. The greatest difficulty was experienced in taking the angle of declination (*see* fig. 3), and in some instances it was necessary to have sections of the bones made to determine it accurately. Any angle of depression between 110°

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[1] It is much to be regretted that Dr. Young does not describe these symptoms *in extenso.*

[2] This is very insufficient evidence of rickets.

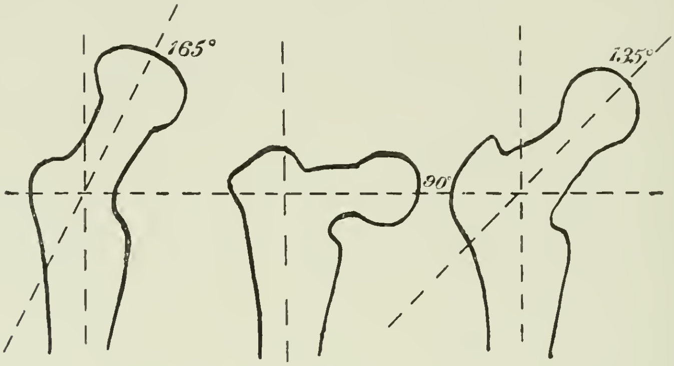

Fig. 4.

Coxa Valga. Coxa Vara. Normal.

Variations in angle of inclination (Young).

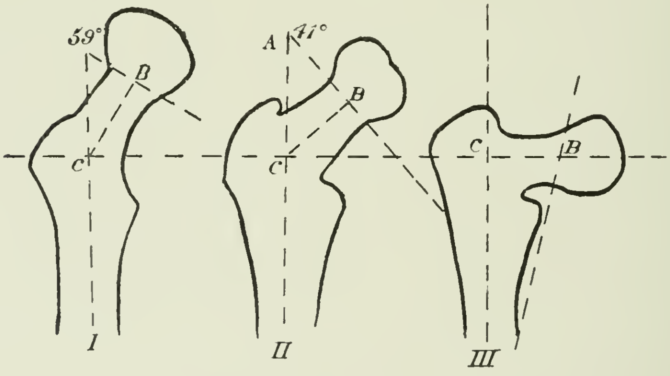

Fig. 5.

Coxa Valga. Normal. Coxa Vara.

Variations in Alsberg's angle.

and 135° was considered normal, and below 110° as coxa vara, and above 140° coxa valga (fig. 4). The length of the femora was considered, but is not recorded here. The number of femora examined at the College of Physicians was 206, and at the Wistar Museum 615, making a total number of 821. In these there were 8 cases of coxa valga, or 0·97 per cent.; 52 coxa vara, or 6·33 per cent., and the remainder were normal, over 75 per cent. of these having an angle of 130°.[1] Of the specimens of coxa valga examined, one was due to tuberculous osteitis, four to arthritis with exostosis, one to rickets, one to hydrocephalus, and in the others the cause could not be determined from the records. The angle varied from 138° to 150°.

The specimen of coxa valga from hydrocephalus, described by Young, is exactly similar to one reported by Humphry in the Cambridge Museum. In that specimen the angle of each neck was 148°; in Young's, 152°. No example of coxa valga from amputation of the femur in early life like those observed by Turner and Humphry was observed. All the eight specimens of coxa valga examined were unilateral except the one due to hydrocephalus.

DESCRIPTION OF SPECIMENS.

By J. K. Young.

No. 1.—"1,174-34-4. Angle, 145°. Arthritis and deformity of head."

No. 2.—"1.175-34-4. Angle, 142°. Upper third of left femur, caries of head and neck. Head and upper part of neck missing.[2] Adjacent surfaces of bone are rough and show more or less periosteal deposit. In the remains of the neck there is a cavity 1·5 cm. deep, but

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[1] Of eleven fœtal skeletons examined at the College of Physicians the development was not sufficient to allow of any exact measurements. The head and neck were "ligamentous," and were usually in the axis of the shaft. The measurements of the angles of the infant skeletons showed the usual high angles recorded by other writers.

Infants: Wistar Museum.

Specimen	4204	3 years	Angle of inclination,		135
,,	4205	1 year	,,	,,	145
,,	4202	11 months	,,	,,	145
..	4203	1 year, 41 days	,,	,,	150
,,	4201	2 weeks	,,	,,	115

[2] If these were missing how could the angle of inclination be accurately determined?—A. H. T.

not showing any sinus. Vertical section shows a healthy shaft and healthy spongy tissue of the trochanter major."

No. 3.—"1,168-35-4. Angle, 142°. Arthritis and exostosis of head. There is no malformation except at the junction of the head and neck, which is marked by a thick and prominent overhanging ridge of bone. At the inner side of the head this ridge is prolonged to the pit for the ligamentum teres."

No. 4.—"1,171-35-4. Angle, 150°. Arthritis and deformity of head. Exostosis and eburnation."

No. 5.—"1,229-34-6. Angle, 150°. Anterior bowing of shaft of bone. Axis taken from upper shaft. Rickets, adult bone, but only 31 cm. in length. Posterior wall thick, anterior wall thin."

No 6.—"430-17. Angle, 150°. Skeleton of negro child, aged 6. Hydrocephalus; bilateral coxa valga. Circumference of head, 70·5 cm. (27·75 in.). The long bones are thin and delicate, and the entire skeleton light in weight."

No. 7.—"2,069. Angle, 145°."

No. 8.—"10,300. Angle, 145°."

"In order to make the examination of the specimens of value every femur in the collection was accurately measured. In this manner it was found that there were fifty-two specimens of coxa vara. Among the 821 femurs, 21 were found among the 206 specimens in the collection of the College of Physicians, which contains mostly pathological material, and 31 were in the Anatomical Institute. The angles[1] varied from 40° to 109°. There were a large number of specimens of impacted fracture of the neck of the femur, with angles below normal, but these were omitted."[2]

Galeazzi, of Milan [7], describes two cases:—

(1) A healthy girl, aged 12, without any history of rickets, but poorly fed, had shown signs of difficulty in walking for a period of two years. There was no history of either traumatism, rheumatism, osteomyelitis or tubercle. She complained of becoming easily tired and of limitation of movement in the joint, and the left hip-joint was painful. Examination showed a characteristic gait in walking, with marked twisting forwards of the pelvis on the left side, which only disappeared when the patient walked with her left leg externally rotated. In an erect posture, and with her legs parallel, it was noted that the normal

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[1] Which angles? A. H. T.

[2] These descriptions leave much to be desired.

lumbar lordosis was increased. The left anterior superior spine was in front of and below the right. She also had slight genu valgum on the left side, and when standing she slightly flexed her left leg, as if it were too long for her. Seen from the front the left hip appeared flatter than the right one. When the patient stood on her left leg only a characteristic phenomenon took place. A marked inclination of the pelvis and of the trunk to the left occurred, that is to say, the reverse of Trendelenburg's sign was produced. In dorsal decubitus, and with the legs parallel, the left anterior superior spine was 12½ cm. from the plane of the bed, and the right 11 cm., and this difference became more marked when attempting to correct the outward rotation of the leg. The distance from the left anterior superior spine to the internal malleolus was 2 cm. greater than on the right side. On passive movements abduction was freer than normal, while adduction and internal rotation were abolished. The left great trochanter was 2 cm. below Nélaton's line. An X-ray examination showed that the head of the femur was in a right line with the diaphysis and only one half of the globular surface of the head, and that the inner and upper half was in contact with the cotyloid cavity (fig. 6). The case was treated by an osteotomy to be described presently, and the angle of inclination was diminished from 160° to 130°, and the angle of Alsberg from 67° to 47°.

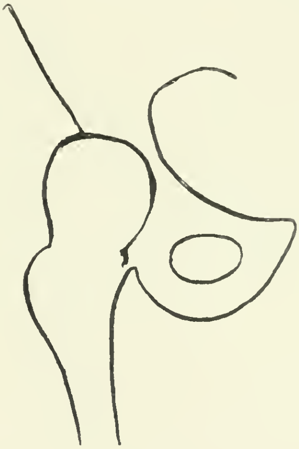

Fig. 6.

Coxa Valga in Galeazzi's first case. Before operation.

In Galeazzi's second case, a young girl, aged 13, showing no rickety signs, came under observation. In this patient the gait had been imperfect since she began to walk, though she had only become noticeably lame four or five months previously, and the left hip had been very painful. In this case there was greater oscillation of the trunk than in the previous one, so that the girl walked with her left leg in abduction, and slightly flexed at the hip, and the trunk was much inclined to the left side. The left leg was 1½ cm. longer than the right, and Nélaton's line was lowered. A certain degree of atrophy in the glutei muscles was noticed, but adduction was the only movement that was limited. The X-ray examination showed an angle of inclination of 169°, and Alsberg's angle measured 94°. The femoral head had lost its roundness, and appeared globular externally (fig. 7). The higher part of the head was not in the acetabulum, and not more than a quarter of the whole head was in contact with the cotyloid cavity. This explains the unsteady gait and the symptoms of luxation in walking.

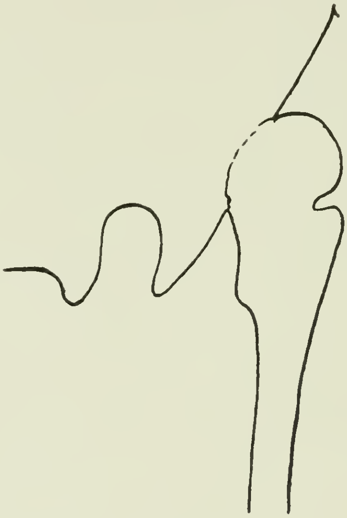

Fig. 7.

Galeazzi's second case of Coxa Valga.

These cases are valuable, as they represent careful observations.

Nathaniel Allison [2] reports the following case of acquired coxa valga: F. S., male, aged 10, first seen in 1904. Hitherto he had been always healthy. In that year he injured his right leg by a fall from a fence, and since then has been lame. The limb was tender for some weeks, and very sensitive. After that he walked on this limb without pain, but with it always abducted and externally rotated. When he was examined in August, 1904, the right hip was abducted to an angle of 60°, flexed to 20°, and externally rotated to 90°, and all movements were limited to a few degrees. There was spasm of the adductor tendons, and an X-ray plate was taken of the right hip, which showed an abnormal condition of the acetabulum, probably the result of a fracture. Under an anæsthetic manipulations were carried out, and the hip was bent at 45° and abducted 20°, but no movements of external or internal rotation were made. The limb was placed in a plaster of Paris spica. After two weeks this was removed, and the walking was painless, but the amount of movement in the hip-joint had not been materially increased by the manipulations. Further treatment was refused for a time, and the boy was taken home.

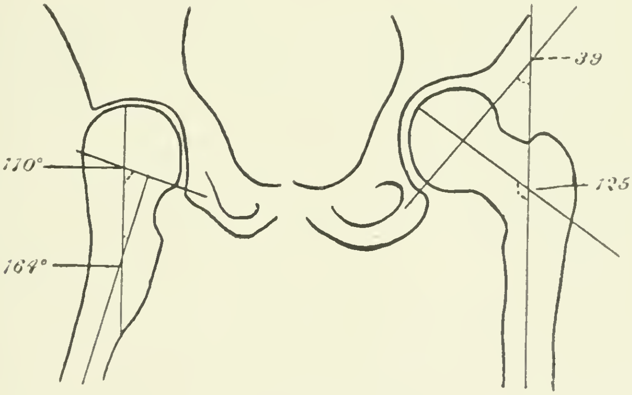

FIG. 8.
Allison's case of Coxa Valga (right) before operation.

In August, 1906, he returned to the hospital for operation. The degree of abduction was then found to be 30°, flexion 15°, and external rotation 90°. The angle of inclination of the right femur was calculated at 164° and of the left at 125° (*see* fig. 8). The length of the right limb, taken from the anterior superior spine to the internal malleolus, was 1 in. greater than the left, and the right great trochanter was depressed 1 in. below Nélaton's line. It was situated below and behind its normal position, and difficult to palpate.[1]

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[1] This observation on the situation of the trochanter points to anteversion of the neck of the femur.

When the boy stands or walks the right leg is abducted and rotated outwards, which causes secondary scoliosis. He cannot bring the feet together nor alter the position of the limb. Passive movement of the right hip is limited to a few degrees in any direction.

The diagnosis was made on the symptoms, on the lengthening of the limb, and the fact that this lengthening "can be accounted for by the increase in the angle of inclination of the femur on the right side." "Treatment consisted in a transverse osteotomy of the femur just below the great trochanter. The lower fragment was rotated inwards 10°, and the thigh adducted 10°, while the flexion of 15° was reduced and the limb was put in plaster of Paris for six weeks. It was then found that the right limb had become shortened by $\frac{7}{8}$ in., the trochanter had been raised $\frac{3}{4}$ in., and the boy stands with the feet together and with no distortion of the trunk." The angle of inclination was reduced to 140° (*see* fig. 9). The anterior superior spines were on a level, and there was no pelvic tilting. Active movement in the hip was not possible, and passive motion was limited to a few degrees. He walks without pain, and the peculiarity of the gait is very much lessened.

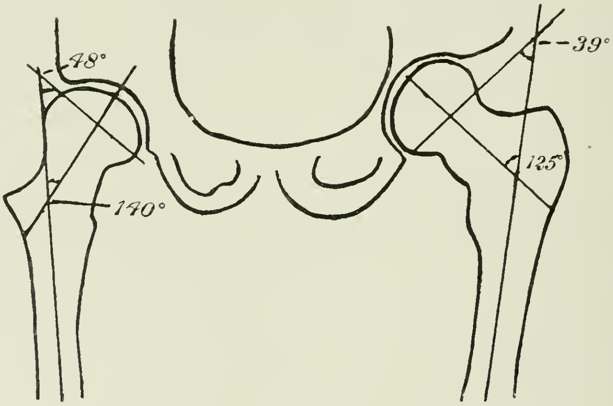

Fig. 9.

Allison's case of Coxa Valga (right) after operation.

Putting aside for the present the pathogenesis, we may deal now with the *causes*. So far as the collected cases go, we may classify them as follows:—

A.—Congenital.

These may be either (*a*) in conjunction with congenital dislocation of the hip, or (*b*) not associated with abnormalities elsewhere.

With regard to (*a*) *Association with Congenital Dislocation of the Hip.*—X-ray photographs of Nos. 1, 2 and 3 of my cases show this quite clearly, and this is a point of great clinical importance, particularly in the treatment of congenital dislocation. Etiologically the question arises whether the coxa valga in these cases is a primary condition of the head of the bone, which has slipped out either before, during or after birth, because so small a portion of it is embraced by the cotyloid cavity; or whether the coxa valga is secondary to the dislocation, and is caused by the outward push of the femur against the head in walking. This outward push must necessarily cause a stress on the epiphysial line of the neck, and so constantly tend to deflect the head upwards. Whatever may be the order of events, the indisputable fact remains that congenital dislocation and coxa valga are sometimes combined; and this affords an explanation of the extreme difficulty, not only in reducing such dislocated heads, but also of retaining them in the cotyloid cavity. The writer strongly suspects that in those cases of dislocation, where the lower limbs must be put in what is called the axillary position, namely, with the knee pointing well upwards towards the corresponding axilla, so as to retain the head of the femur secure, coxa valga is present in a considerable degree. The following cases have come under my notice:—

Case I. Congenital Dislocation of the Hip.—G. M., aged 2¼, came to the Royal National Orthopædic Hospital in October, 1905. It was noted that she halted on the left hip, and that the left leg was smaller than the right, especially above the knee. As she stands the body is deviated towards the left side, the left trochanter is prominent, and the head of the femur is displaced upwards. There is no scoliosis. Trendelenburg's sign is present. This is due to dislocation of the hip, which masks the sign of coxa valga in this respect. The limb is externally rotated; it is not abducted. All the movements of the limb are very free.

MEASUREMENTS.

	Right	Left
Umbilicus to internal malleolus	17½ in.	16½ in.
Anterior superior spine to internal malleolus	16⅜ in.	15¾ in.
Nélaton's line	Normal	Left trochanter 1¼ in. above

A skiagram taken with both feet slightly everted shows the dislocation of the left hip, and the angle of inclination on that side to be 169° and on the right side 126° (fig. 10).

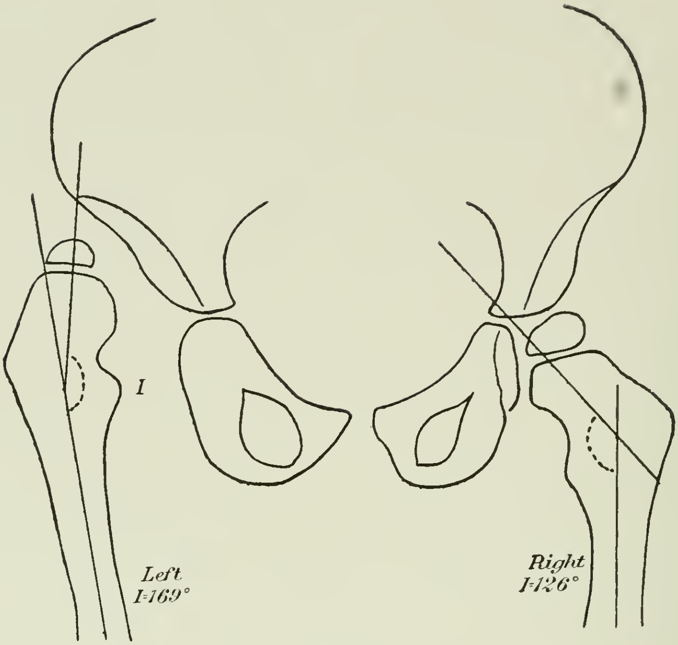

Fig. 10.

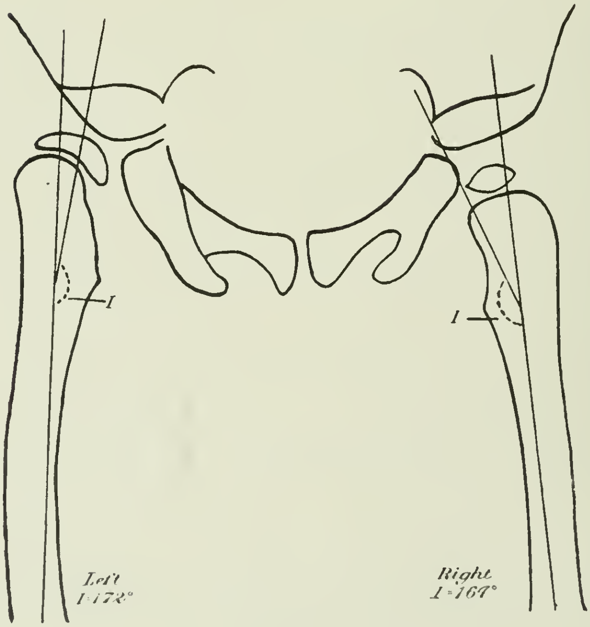

Fig. 11.

Case II. Congenital Dislocation of the Left Hip, with Coxa Valga.—L. M., aged twelve months, was admitted to the Royal National Orthopædic Hospital on January 21, 1907, for congenital dislocation of the left hip. It is a striking fact that he is the second child of a family of three, who are all under my care for congenital dislocation of the left hip. The first and third children are females. In this case the left hip was reduced by manipulation and put up in plaster. On October 25, 1907, an X-ray photograph was taken, from which the diagrams are made. The head of the left hip was seen to be in the acetabulum. The angles of inclination were as follows: On the left side 172°, on the right side 164°, taken with the legs lying lax (fig. 11). The great increase in the angle of inclination on the left side shows that in this patient, and probably in the children of this family, the upper end of the femur is of such a shape as to cause the head of the thigh bone to be only partially and insecurely placed in the acetabulum, and therefore liable to slip out under the slightest provocation.

Case III. Congenital Dislocation of the Left Hip, with Coxa Valga.—H. S., aged 2, was admitted to the Evelina Hospital under my care on October 29, 1903, with congenital dislocation of the left hip. There was ¼ in. shortening on the left side, and the movements of flexion, abduction and adduction were limited. On December 1, 1903, the Hoffa-Lorenz open operation was performed on the left hip. The capsule of the joint was ⅛ in. to a ¼ in. thick. This was opened by a crucial incision. At the same time particular care was taken to freely divide the lower part of the capsule and the ilio-psoas tendon. The acetabulum was found to be of good shape and normal. After division of the above structures, no difficulty was found in placing the head of the bone in the socket so long as the limb was abducted to an angle of 40°.

When seen on December 15, 1904, the legs were equal in length; she could flex the limb to 45°; abduction was excellent, adduction was limited, and extension was full. There was no telescoping of the head of the femur, and it appeared to be in good position and stable; the foot was not everted.

On June 22, 1905, the head of the femur was still in good position and she was walking well. An X-ray photograph was taken on January 26, 1907. The head of the left femur is seen to be thoroughly well placed in the acetabulum, but the upper and posterior part of the rim of that acetabulum is not so well defined as the right, and about a third of the sphere of the head of the femur is not embraced by the cotyloid cavity. The angle of inclination of the left femur is 154° and of the right 147° (fig. 12).

This case is interesting as it is one of the three successful cases in which I have been able to reduce the head of the bone by the open operation, and in this case there has been no recurrence of the dislocation for four and a half years. I have the pleasure of demonstrating the cases of G. M. and H. S.

(b) Coxa Valga, not associated with other Abnormalities.—Young is responsible for this subdivision of the congenital variety in which no other abnormalities are present. While it was stated that any increase in the angle of inclination of the femur above 128° constituted coxa valga, yet physiologically the angle may be increased to 140° without definite symptoms following. There is, however, one case of coxa valga, published by David, which is assumed to have been congenital, but the evidence is not conclusive. Galeazzi has observed that the normal direction of the epiphysial line of the neck is marked by a line parallel to the aperture of the cotyloid cavity. It is probable, therefore, that careful observations on fœtuses and on the newly born will discover some cases of this type of congenital variety.

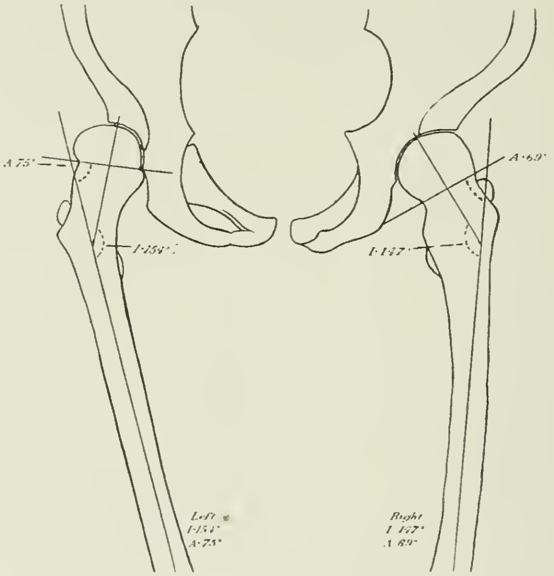

Fig. 12.

B.—Acquired.

(1) *Due to Traction Exerted by a Pendent Limb.*—It is readily conceivable that the compound effects of continuous traction by the weight of the part, the absence of body weight above, and the loss of the normal strong contractions of the pelvi-femoral and pelvi-trochanteric muscles may lead to coxa valga. Such conditions are more or less met with in all cases of infantile paralysis, and even when inactivity of the limb is very marked. It is possible that coxa valga would be more frequently seen in association with paralytic limbs which are incapable of weight bearing were it not for the fact that, owing to the capabilities of modern orthopædic apparatus, paralytic limbs can be made to bear some proportion of the body weight.

Specimens and cases of coxa valga in infantile paralysis of the lower extremity have been described and recorded by Humphry, Turner, Lauenstein, Albert, and Young; and I am able to give a description of a case and show a patient who came under my observation at Westminster Hospital.

Case IV. Coxa Valga on the Right Side, associated with Infantile Paralysis.—P. S., aged 9, came under my care at Westminster Hospital on November 15, 1907, on account of infantile paralysis of the right leg, which had existed since he was 2 years of age. It was seen on examination that there was extreme atrophy of the glutei muscles, of the thigh and of the leg. There were no congenital defects and no history of injury, rickets, or tubercle. The patient was an anæmic and under-fed boy. He cannot walk without support, but when he uses crutches the right thigh is flexed on the abdomen to an angle of 40°, and the leg is flexed on the thigh at an angle of 30°, while the foot is extended and everted, and the back shows a double scoliotic curve, the dorsal deviation being to the left and the lumbar to the right. There is also considerable lordosis. The right side of the pelvis is twisted so that the right anterior superior spine is on a plane lower and posterior to the left. There is also some genu valgum on the right side, and some contraction of the tensor vaginæ femoris and ilio-tibial band is noticeable. The electrical reactions show the reaction of degeneration in all the muscles of the right leg, and the patient has no voluntary power of movement. As he lies in bed the right limb is flexed at the thigh and knee and falls inwards. Measurements were taken of the limbs:—

	Right	Left
Anterior superior spine to knee	11⅞ in. ...	12½ in.
Knee to external malleolus . ..	9½ in. ..	10¼ in.
Great trochanter	½ in. above Nelaton's line ...	Normal

On passive movements of the limbs it was noted that the movements of the right hip were unusually free, except when limited by the contracted ilio-tibial band. The thigh could be fully flexed, and when it was rotated downwards and outwards through a wide area the head of the bone was felt to slip upwards as if subluxated. After this date (February 3) this sign disappeared, and the patient has been resting in bed with weight extension on, and a good deal of the tension of the contracted ilio-tibial band has been removed. The right trochanter is about ½ in. posterior to its normal position, and its external aspect is rotated somewhat backwards. Trendelenburg's sign, or its reverse, is not obtainable, as the patient cannot stand. X-ray photographs of the limbs were taken in the following positions:—

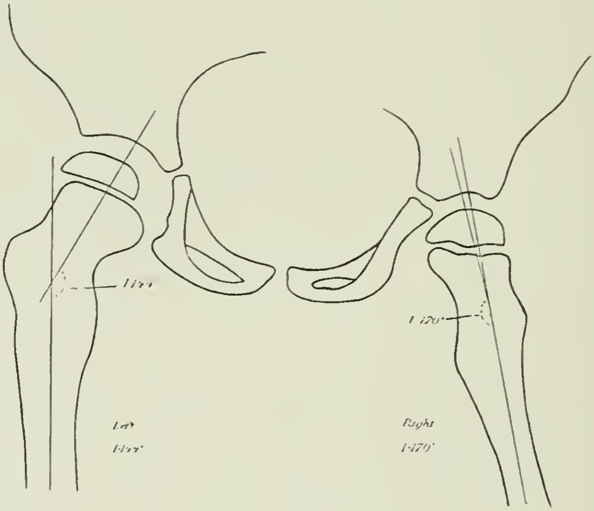

Fig. 13.

(*a*) As the patient lay upon the couch, that is, with the right foot everted at an angle of about 45°, the angle of inclination on the right side was found to be 170° and on the left 144° (fig. 13).

(*b*) With both feet placed parallel the angle of inclination was found to be 150° on the right side and on the left 143° (fig. 14).

(*c*) With both feet fully everted and the limbs abducted, the angle of inclination on the right side was 204° and on the left 160° (fig. 15).

(*d*) With both feet fully inverted the angle of inclination of the right appeared to be 128° and of the left 133° (fig. 16). That is to say on the right side, between the fully inverted and the fully everted positions, there were 76° of difference; and on the left 27° of difference. This evidences not only the possibilities of fallacy in examining these cases unless X-ray photographs are taken in all positions, but also the extraordinary elevation and twisting of the neck which must have taken place in the right femur to allow of this rotation through such a wide arc as 76°.

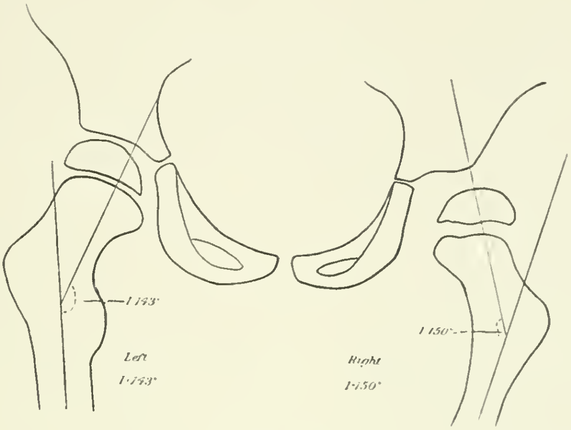

Fig. 14.

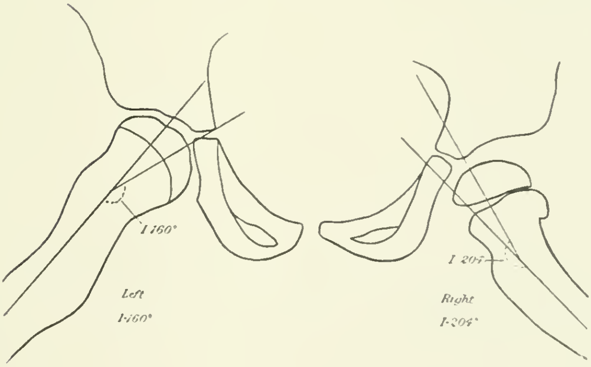

Fig. 15.

With regard to the future treatment, section of the contracted band of fascia will be done and the boy supplied with an orthopædic apparatus, taking its bearing from the ischial tuberosity, and made rigid from the hip downwards. It is useless to attempt any other treatment for this limb except persistent massage and galvanism. If recovery of any of the muscles takes place the possibility of tendon grafting and arthrodesis may be considered.

Coxa valga also occurs after amputation through the thigh in early childhood, as we have mentioned above.

(2) *Static or Functional Varieties.*—Wolff's law shows that the shape of the bones is determined by conditions of reaction to the body weight and the stress and strain of muscular action. A femur performing its functions in an adducted position will eventually assume a different shape to one which works in an abducted position, so that in tilting of the pelvis differences in the shape of the thigh bones are to be anticipated. When the centre of gravity of the trunk is disturbed, as in scoliosis, the femora will be subjected to different conditions of stress and strain due to unequal loading. Unloading or loss of the normal load is just as much a static condition as overloading. It is, as it were, a "negative phase." Certain facts show that considerations of this nature must be taken into account in seeking to explain some cases of coxa valga.

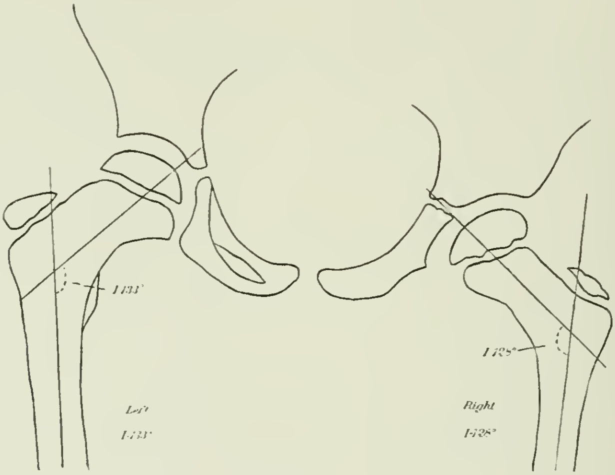

Fig. 16.

In hip-joint disease, where the limb has been abducted, Galeazzi says that a degree of coxa valga has been induced. Unequal loading perhaps explains the coxa valga in the scoliosis case of Mauclaire and in the genu valgum case of Gangolphe. It is highly probable that static conditions govern the association of coxa valga and genu valgum noted by other writers. Thus genu valgum on one side is associated with coxa vara on the same side and coxa valga on the other. In Galeazzi's case, in which the patient always leaned on a paralytic limb in walking, the pelvis became tilted downwards on that side, so that the limb was in an abducted position, and coxa valga resulted. If in this case the femoral neck had been deficient in resistance or incapable of reacting to the stimulus of this increased strain, it is probable that it would have collapsed and coxa vara resulted.

A case of what may be termed compensatory coxa valga from fracture of the shaft of the femur, which I am enabled to show you, came also under my notice at the Royal National Orthopædic Hospital. The description of it is as follows :—

Case V. Right Coxa Valga, following Fracture and Malunion of the Shaft of the Femur.—G. M., aged 8, came under my observation at the Royal National Orthopædic Hospital on July 18, 1907, on account of lameness in the right leg. In October, 1906, he was run over by a motor-car and was taken to Hackney Union Infirmary. The right thigh was considerably crushed, and he remained in the infirmary until Christmas. The parents said that when he left the infirmary there was no support placed on the leg. On account of the lameness he went again to the infirmary last Easter. He was placed in bed for a time, and then went to a convalescent home. On examination the child is a pale, under-sized boy, and stands with the right leg bowed out, and there is an extensive curve outwards occupying the greater part of the right femur. The right trochanter has lost its natural prominence, and in this situation there is a marked depression. The external surface of the trochanter looks backwards. There is no scoliosis. Trendelenburg's sign is reversed. When he walks the chief sign is limping, but he has no pain. The limb itself is rotated outwardly, but abduction is not marked. When he lies down flexion at the right hip is normal. Abduction can be carried out to an angle of 45°, adduction is limited to 20°, external rotation to 30°, and internal rotation is free only when the thigh is flexed.

MEASUREMENTS.

	Right		Left
Umbilicus to internal malleolus	27 in.	...	27½ in.
Anterior superior spine to internal malleolus...	24 in.	...	25 in.
Nélaton's line	Right trochanter normal	...	Left trochanter ½ in. above

An X-ray photograph was taken of the right leg with the foot to the front, and the angle of inclination was found to be 152° (fig. 17).

With regard to the treatment the obvious thing to be done is to remedy the mal-union of the femur by operation. When the static conditions are altered it is almost certain that at his age the coxa valga will gradually disappear.

The connection between genu valgum and coxa valga is illustrated by the following case, which came under my notice at Westminster Hospital, on December 6, 1907.

Case VI.—C. M., aged 11, was admitted for "malformation of both hips." The only point in the history is that there is a difficulty in progression since the child began to walk. There was no history of tubercle, injury, or paralysis, and no congenital defects are noticeable. No history of rickets. The patient is a feeble boy, and stands and walks on a wide base, with feet apart and the knees approximated. Both feet are flattened and the knees are valgoid. There is slight scoliosis, with the convexity of the lumbar curve to the left. On walking he rolls about, but does not limp, the pelvis drops to the left side and there is slight lordosis. It is seen that the right trochanter is not so prominent

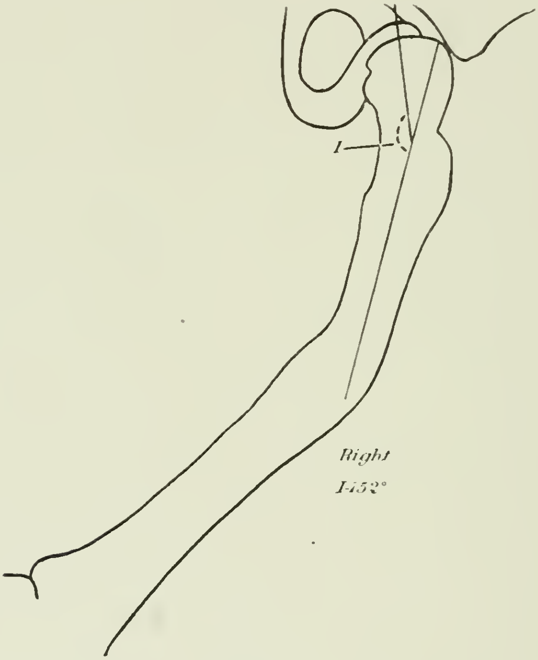

FIG. 17.

as the left. The left leg shows external rotation at an angle of 45° on standing, and the distance between the malleoli, with the knees ⅛ in. apart, is, when he stands, 5½ in., and when he lies down 4½ in. The measurements are as follows :—

	Right		Left
Umbilicus to internal malleolus	25⅝ in.	...	25⅝ in.
Anterior superior spine to internal malleolus	24¾ in.	...	24¾ in.
Trochanter	⅛ in. above Nélaton's line	...	Normal, but displaced slightly backwards

Trendelenburg's sign is absent on both sides as they are tested alternately.

Passive movements: with the limbs extended, on the right side, abduction is 40°, adduction 20°; left side, abduction is free to the right angle, adduction is limited to 5°. With the thighs and knees flexed: on the right side abduction is very free and adduction is good; on the left side abduction is very good and adduction is limited. On fully flexing both thighs simultaneously the femora are rotated outwards. It was also noted that the head of the femur on the right side appeared to be unduly movable in the acetabulum.

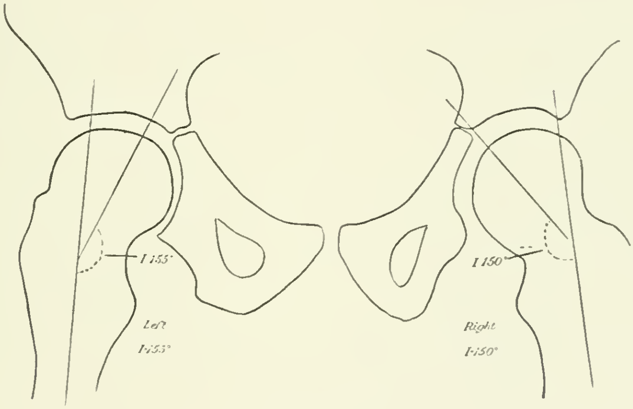

Fig. 18.

Skiagrams were taken in three positions: first, with the patient lying as he was accustomed to do, the angle of inclination on the right side was found to be 150°, on the left side 155° (fig. 18). The feet were then fully inverted, and the angle on the right side was 130°, on the left 137° (fig. 19). The limbs were then fully turned out; the angle on the right side was 163°, on the left 170° (fig. 20).

With reference to the treatment of this case, the right thing appears to be to cure the genu valgum and to note the effect on the angles of inclination.

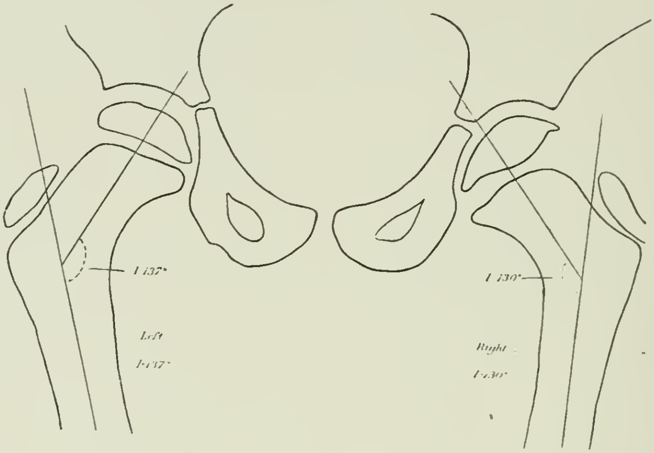

Fig. 19.

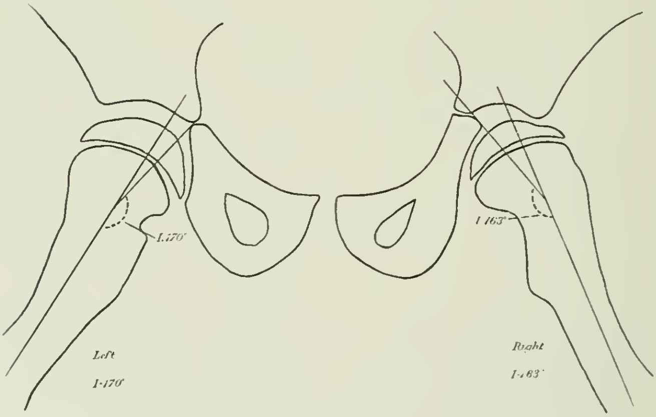

Fig. 20.

(3) *Traumatic.*—The deformity arises after fracture of the neck of the femur, with impaction and mal-union, and after injuries and separation of the epiphysis. It is rare, but it is quite conceivable, that it can be produced by a direct fall or blow on the great trochanter. Thiem [20], Hoffa [10], Kölliker [13], Manz [16], J. K. Young [24] and

Drehmann [6] have recorded cases. The case quoted by the last-mentioned writer is somewhat striking. A woman, aged 46, fell on her right hip. Although it was very painful she was able to walk a considerable distance to her home. The leg was abducted and lengthened. In a few days she was about again, using a stick. The movements were considerably interfered with, especially those of adduction and rotation inwards. Later on the injured limb was found to be 2½ cm. longer than the other, but it was also much abducted, and it was found that the lengthening could not be neutralised by tilting the pelvis upwards on the injured side. A high boot, with a thickening of 3 cm. to the sole and heel, was fitted to the sound side. A skiagram showed the existence of an impacted fracture, coxa valga, and an angle of inclination of the neck of 155°. This case, then, affords one interpretation of a fact noticed in general surgical text-books, that occasionally actual lengthening may result from fractures in the neighbourhood of the hip-joint.

Bradford and Lovett [4] also state that coxa valga has been recorded following a severe fracture of the lower end of the femur and knee-joint.

(4) *Associated with Rickets or other Processes of Bony Softening, such as Osteomyelitis and Tubercle.*—Authors speak of a rhachitic form of coxa valga, and undoubtedly some instances are met with in rickets, though primary rickety coxa valga is probably rare and the affection is more or less a secondary result. It is well known that if the neck of the femur is soft it will bend downward, and coxa vara, not coxa valga, will result. What is actually seen in some cases of rickets is an inward bend of the lower part of the shaft of the femur, as in my case of fracture; while the femoral head is abnormally in a line with the upper part of the curved shaft, that is, a compensatory coxa valga has developed, neutralising more or less the primary rhachitic deformity. Tilanus [21] gives details of and figures a case of compensatory coxa valga, occurring in a boy, aged 12, in the course of infantile osteomalacia.

(5) *Idiopathic.*—This term is used because certain cases, not readily falling into the above groups, are met with, and have been considered to be analogous to a form of coxa vara. The latter is really a static condition, and it is very probable that the so-called idiopathic cases of coxa vara depend upon an anomaly of gait or attitude. Other cases are probably congenital. To the author, at all events, an idiopathic coxa valga does not appear probable, except possibly in osteomyelitic softening of the neck of the femur, with treatment by weight extension. It is

possible, however, that cases may be met with due to some abnormality in the growth of the bones or associated with a wedge-shaped epiphysial cartilage at the neck of the femur, the base of the wedge being downwards. Even if such cases occur, it must be a very fine line which divides such forms from congenital ones. Again, it must not be forgotten that in the fœtus coxa valga is a normal condition, and may persist after birth.

Pathogenesis.—Much light is thrown upon this point by the remarks on causation, but a few words may be added. The normal shape and position of the head and neck of the femur depend upon the nice adjustment of certain forces, namely, weight pressure, resistance of the bone to this pressure, and muscular effort. If any one of these factors, or all of them, are varied in degree, altered in direction, or destroyed, changes take place conformably with Wolff's law, and the head and neck of the femur assume different positions and directions. To take an example from the better-known deformity, coxa vara, if the resistance of the bone is lessened by rickets, the effects of the superincumbent weight and the pull of muscles come into play, and we find the head of the bone gradually sinking until it is at a right angle or less. Again, if the continuity or the resistance of the neck be suddenly destroyed by separation of the epiphysis or fracture, the pelvi-femoral and pelvi-trochanteric muscles retract to their fullest extent, drawing the shaft of the femur upwards, and coxa vara results. That is to say, in these traumatic cases an alteration in one factor only, namely, the tensile strength of the neck of the femur, results in coxa vara. Now let us suppose that the weight of the body above or—what comes to the same thing—diminution of pressure from below is brought about, that is, the limb is pendulous. Then the most important factor in determining the normal angle of the neck of the femur is lost, and as it grows it approximates towards a straight line with the shaft. It must also not be forgotten, in connection with infantile paralysis, that the pull of the powerful psoas muscle, which curves beneath the neck of the femur, and is responsible to some degree for the normal angle of the neck, is either much diminished or entirely destroyed. There is no doubt that the normal ilio-psoas is a factor of importance in the production of the usual angle, and when the muscular force is lost the angle tends to increase.

In the treatment of fracture of the neck of the femur, when extension by weights is used, great care should be exercised that the limbs are placed parallel and that the proper amount of weight is employed.

If too much, the shaft will be dragged too far downwards, and the head will unite at a very obtuse angle; coxa valga will be produced. If too little weight is used, the opposite condition, coxa vara, will follow. Therefore every case should be examined through the screen from time to time after it has been put up, so as to ascertain that the fracture is in the best possible position.

In congenital dislocation, when the head is displaced upwards, the thrust of the pelvis downwards and outwards in walking in unilateral cases must naturally cause the angle of inclination to open out, and the effect of this thrust is increased by the frequently abducted position of the limb. In scoliosis there is no doubt that coxa valga arises on that side to which the pelvis inclines, but further observations are needed by means of the X-rays to ascertain if coxa vara on the opposite side is always associated.

As to the occurrence of coxa valga in rickets, it is difficult to explain why it arises. The opposite deformity, coxa vara, is easily explicable and rational, but, as we have already remarked above, rickety coxa valga must be excessively rare. When it occurs secondarily to some other deformity, such as genu valgum, static conditions may afford the necessary clue.

Symptoms and Signs.—The cardinal symptoms are abduction of one or both lower extremities, associated with external rotation and limitation of adduction.

(1) *Pain and Spasm.*—At the onset of the trouble the patient often experiences fatigue and wandering pains in and about the hip-joints, and sometimes it will be noted that the abductor muscles of the limbs are in spasm.

(2) *Gait.*—In a unilateral case it is limping, and the trunk is inclined towards the affected side. In bilateral cases it is rolling, sailor-like and unsteady, and is reminiscent of the walk in congenital dislocation. The cause of the unsteadiness and uncertainty is that the head of the femur is not firmly and wholly planted in the acetabulum. Only the inner and upper portion of the convexity of the sphere lies in the cotyloid cavity. In some cases a partial subluxation occurs in walking; or when the patient is lying supine and the limb is flexed, abducted and rotated outwards, a distinct slip upwards of the head may be felt.

(3) *Lengthening of the Limb.*—In a unilateral case the limb is lengthened 2 cm. to 3 cm., and tilting upwards of the pelvis does not compensate for the increased length. In fact, it may be necessary, in order to render the limbs parallel, to compensate on the sound side.

(4) The limb is abducted and rotated out, while movements of adduction and inward rotation are limited. This is said to be pathognomonic. There is often some slight limitation of flexion.

(5) In bilateral cases the patient stands on one leg only with difficulty. In unilateral cases, when the patient stands on the affected limb, the body is inclined towards the affected side. This is well shown in the photographs of Galeazzi's case [8]. Now this posture is just the opposite of that which obtains in Trendelenburg's sign, which is often seen in congenital dislocation of the hip, where, when the patient stands on the affected limb, the body drops to the opposite side.

(6) Compensatory changes occur in the trunk. In unilateral cases the upper part of the body is inclined towards the affected side, and there is lumbar scoliosis with its convexity towards the affected limb. Sometimes the pelvis is twisted, so that the anterior superior spine on the affected side is below and in front of the opposite one.

(7) The region over the great trochanter is often flattened. This is in great contrast to what is seen in congenital dislocation, where it is unduly prominent. Further, the great trochanter is often below Nélaton's line, and its outer surface is frequently displaced backwards. This displacement backwards points to the probable existence in coxa valga, not only of increase in the angle of inclination of the femur, but also of incurvation, or a forward convexity of the neck, as in coxa vara. There may also exist some torsion of the neck on itself, but no observations exist on this point. The lowering of the great trochanter is not observed if there be a widened acetabulum, or if the case be complicated by congenital dislocation of the hip, or be associated with infantile paralysis.

(8) Finally the last word rests with careful skiagraphy, and we think that all cases described as coxa valga will not bear close analysis. Skiagraphy requires great care, and the position of the limb must be carefully noted. Thus a case of coxa vara, if rotated out through 90°, will appear as coxa valga, that is to say the further we deviate from a plane transecting the femoral neck frontally, the more valgoid will the neck appear. There are, however, two means of avoiding this failure. We should be certain that we see the anterior edge of both trochanters clearly in the skiagram. A more reliable method is that which I have adopted of having the X-ray photographs taken in at least three positions of the limbs, namely, both feet fully inverted, both feet straight to the front, and both feet fully everted. If the appearance of coxa valga is seen in all three positions, and if the measurements of the angle of

inclination are in each instance excessive, then I take it that there is no doubt of its existence. An excellent method is to take the X-ray photographs stereoscopically, and then have a reduction made.

As the cases which came before my notice increased in number, I became aware of the necessity of taking the photographs of the limbs in the positions of eversion, inversion, and with the feet to the front. It is necessary that the tube be kept at each exposure in the same position, and the patient's body as well. Not only should the position of the limbs be specified, but the angle of inversion and eversion noted. In the case of young children it is also necessary to give an anæsthetic.

Many of the records in literature, so far as the angles of deviation are concerned, are vitiated by the absence of the above precautions, and I suspect that, relying too much on imperfect X-ray work, some cases have been designated coxa valga which are not so.

Diagnosis.—The cardinal and other symptoms have been already dwelt upon, but in the various types of cases some of the signs may be modified. Shortening of the limb, rather than lengthening, exists in congenital dislocation and in infantile paralysis due to general atrophy. Again the movements of adduction may be very free in paralytic cases. Further the trochanter is raised, and not lowered, when coxa valga is associated with congenital dislocation, and here, too, the trochanter is also prominent. In some cases the trochanter is displaced backwards, and incurvation of the neck is probably present.

The chief cause of error is to confuse an incipient tuberculous coxitis with coxa valga, but this can be readily avoided if we remember that in the latter upward tilting of the pelvis does not compensate for the lengthening of the limb. Again, sacro-iliac disease, on account of the abducted position of the limb and apparent lengthening and dropping of one side of the pelvis, may lead to mistakes.

Treatment.—The subject has hitherto received such scanty notice that no definite lines of treatment have been laid down. Independently the writer treated one case, and he found afterwards that the same plan had been adopted by David, namely, placing the limb as far as possible in the adducted position. The limb was adducted and rotated inwardly under an anæsthetic, and fixed in a plaster of Paris spica. David succeeded in improving the angle of depression in his case by 7°, but I cannot say that my efforts met with success. In some cases equalisation of the limbs by the use of a high sole on the sound side will not only improve the gait, but will lessen the angle of inclination by changing the direction of the weight pressure.

Wallace Blanchard [3] devised a cuneiform osteotomy of the neck for this condition, but this is not an easy performance, and the success of the after treatment is more problematical than in subtrochanteric osteotomy.

Arguing from the analogy of coxa vara, where the deformity is readily remedied by the removal of a wedge of bone with its base outwards from the subtrochanteric region, it ought to be the right thing to remove in a case of coxa valga the wedge of bone, but with its base inwards. This, however, is a troublesome proceeding on account of the immediate neighbourhood of large vessels, particularly of the internal circumflex and superior perforating arteries.

Galeazzi has obtained good results in his cases by performing a linear osteotomy well outside the joint, at the base of the neck, and not through it. He is convinced that in all anomalies of direction of the neck, the part to be operated upon must be this region, and not the subtrochanteric. He further points out that if the section is made at the spot indicated, the pull of the great muscular masses whose tendons are inserted into the great trochanter is such as to drag the shaft of the femur upwards and lessen the angle of inclination. He afterwards applied sufficient traction to the leg to prevent great displacement of the fragments without preventing the gradual ascent of the trochanter, and he followed step by step by means of X-ray photographs this upward movement of the trochanter, which he stopped at the proper moment by means of a firm plaster of Paris spica bandage. The success of his treatment is indicated by figures (figs. 21 and 22).

In the first case the angle of inclination diminished from 160° to 130°, and the angle of Alsberg from 67° to 47°. In the second case the angle of inclination decreased from 169° to 130°, and the angle of Alsberg from 94° to 40° (*see* fig. 23), the osteotomy having been completely extra-articular. The hip-joints maintained their perfect mobility, the gait became normal, and is still unaltered one year after the operations were performed.

Nathaniel Allison divided the bone just below the great trochanter in his case, and remedied the external rotation and abduction, with the result that the angle of inclination was diminished from 164° to 140°. On account of the original injury very little increase of movement of the hip-joint followed.

I have not operated for this condition yet, but, provided that, as in hospital, I can carefully watch the subsequent progress of the case by means of the X-rays, I should give the preference to Galeazzi's method;

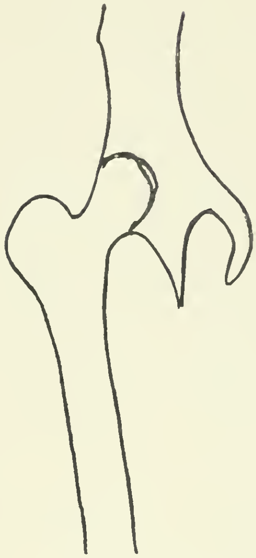

Fig. 21.

Galeazzi's first case, after operation.

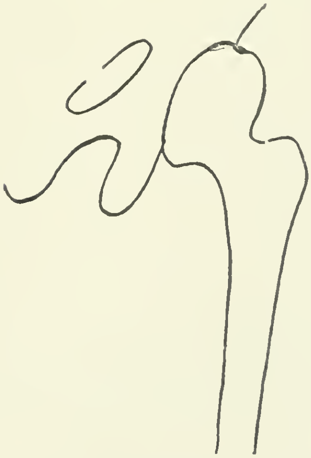

Fig. 22.

Galeazzi's second case, after operation.

whereas under other conditions I am disposed to think that simple subtrochanteric osteotomy, with adduction of the lower fragment of the femur, will suffice. It will not be necessary to remove a wedge, as the gap left in the bone outside will readily fill up with callus.

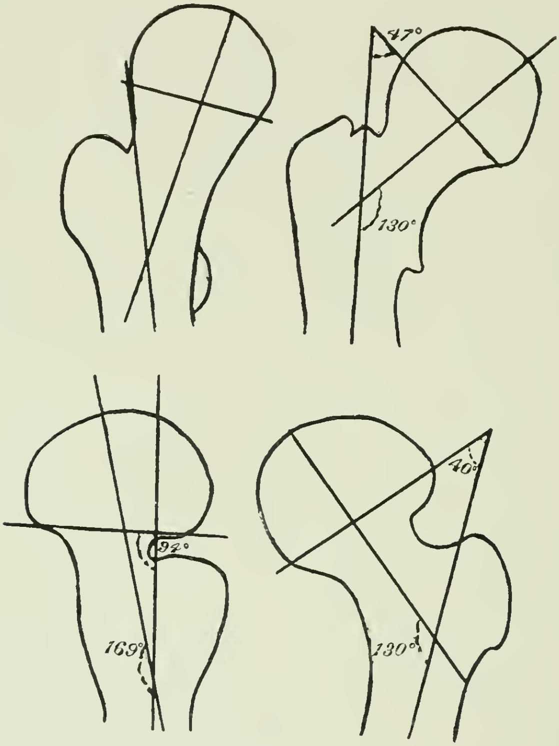

Fig. 23.

To illustrate improvement in Galeazzi's cases.

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DISCUSSION.

Mr. Gordon Watson said it was not clear from the paper what evidence there was of the presence of coxa valga in congenital dislocation of the hip. As the author clearly pointed out the angle of inclination, as seen by the X-rays, varied with the position of the limb. He asked whether he had any actual pathological specimens of femora showing coxa valga in congenital dislocation of the hip. Drawing No. 10 seemed to be an ordinary condition of affairs seen by X-rays in congenital dislocation of the hip. With the marked inversion it was almost impossible to get a smaller angle of inclination appearing on the skiagram. And Mr. Watson was not clear as to what cases came into the category of requiring treatment for coxa valga. Limbs that had never borne weight, such as the flail limbs of infantile paralysis, showed a coxa valga, and an excellent specimen of this variety might be seen in St. Bartholomew's Hospital Museum, showing an angle of about 150°; but these did not call for surgical treatment any more than did amputation stumps with coxa valga. Mr. Tubby had said that the evidence as to coxa valga in rickets was slight, but he had referred to cases, and he asked the author whether there were any specimens of that extant. Mr. Watson could not conceive how the weight of the body, acting through the head of the bone, could straighten out the neck where the bone was soft, even though there was an outward bend of the shaft which required compensation. The mechanism of coxa vara deformity in rickets was obvious.

The President (Mr. Warrington Haward) said it would be interesting to learn whether any other Fellows had knowledge of specimens bearing on the point raised by Mr. Gordon Watson. He confessed that he had been himself unable to see how, in the case of a soft rickety bone, the weight of the body could open out the angle at which the head of the bone was placed in relation with the shaft.

Mr. TUBBY, in reply, said he had no specimens in his possession. As to the evidence of congenital dislocation, he said that the X-ray picture was taken in one position, but that was the position in which the limbs were used for walking, and if the rays showed coxa valga in that position it was probably the position assumed by the femur usually. He had been most careful to assert in his paper, and now repeated the statement, that rickets was not a cause of primary coxa valga. His point was, that as the shaft of the femur came towards the middle line, it was probable that by compensation the angle of the neck of the femur was increased, so that the power might be transmitted more directly from the body towards the feet. The explanation of the secondary coxa valga was an adaptation, so as to allow the weight of the body on that side to be transmitted more directly through the line of the lower extremity. Hoffmeister's case, and that of his boy with genu valgum, showed, by X-ray photographs taken in three positions, viz., with the feet inverted, everted and to the front, that the angle of the neck of the femur was increased.

Surgical Section.

March 10, 1908.

Mr. J. Warrington Haward, President of the Section, in the Chair.

On the Possible Uses of Lumbar Puncture in the Treatment of Otitic Meningitis.

By Arthur E. Barker, F.R.C.S.

Two questions of interest, and perhaps of importance, are suggested, I venture to think, by the three cases which follow. The first is whether surgeons may not with advantage, as the physicians have already done, make use of lumbar puncture for diagnostic purposes more frequently than at present in the early stages of those obscure cases of meningitis due to ear disease with which we are all familiar, and as to which we find it often so difficult to formulate a prompt line of action. The second is whether, when the inflammatory products of meningitis are clearly found by puncture of the lumbar sac, we may not extend the use of the puncture further and employ it as a *remedial* agent for the *repeated* removal of inflammatory fluid from the spinal canal, and consequently to a certain extent from the cranial cavity; for it is almost an established fact that when the spinal canal is relieved of its liquor this is replaced from the arachnoid space round the brain, to which physiologists tell us it is chiefly, if not entirely, furnished by the choroid plexus. By tapping it, then, we should, as it were, flush the cranial and spinal dura repeatedly as well as relieve intracranial tension.

In these two questions physicians and surgeons have a like interest and it is to be hoped that in regard to them the former may now be led to give us their opinion. All of the three following cases illustrate beyond all doubt the value of lumbar puncture for diagnostic purposes.

But, in addition to this, the first shows unmistakably its usefulness as an aid to treatment. The second shows the same, though in a lesser degree; the third illustrates the diagnostic value of lumbar puncture certainly in such cases, and perhaps its remedial benefit to some extent.

A good deal has been done within the last eighteen years, since Quincke introduced the procedure in 1890, in the direction of utilizing the tapping of the lumbar sac for diagnostic purposes in medical cases. And in all the more recent epidemics of cerebrospinal meningitis, for instance, it has been largely employed, and with much advantage. This disease has been at once recognized by the bacteriological examination of the fluid withdrawn and the discovery in it of the meningococcus of Weichselbaum with other leucocytic findings. But besides its use for diagnostic purposes in this disease something has also been done to demonstrate its value in treatment. Several physicians have placed on record (see below) series of cases of epidemic cerebrospinal meningitis treated by repeated tapping of the inflammatory products from the lumbar sac, and by this alone, with a high percentage of recovery.

Lenharz, of Hamburg, appears to have been the first, in 1905, to demonstrate by a series of interesting cases the benefit of *repeated* and *frequent* lumbar puncture at *regular* intervals in the treatment of the epidemic form of the disease. Vorschutz has also published five severe cases, accompanied by torpor and delirium, in which recovery followed this treatment in four, the meningococcus having been demonstrated in all. Bókay had seventeen cases in which this treatment was employed, with recovery in ten. Laurens has also published one successful case, and also Widal, who makes some specially interesting observations in his report thereon. Other series are also referred to in Bókay's paper with very encouraging results (see below). These were all severe cases of cerebrospinal meningitis of the epidemic form in which Weichselbaum's bacillus was found—*Diplococcus intracellularis.*

With these facts before us we are, I think, justified in the hope that in other non-epidemic forms of meningitis, such as those following ear disease, we may possibly find at least a useful adjunct to other means of treatment in repeated lumbar tapping at regular intervals. For obvious reasons continuous drainage of the inflamed spinal dura is out of the question. But, of course, there are other varieties of severe meningitis, such as those due to virulent streptococcal infection, where but little can be hoped for from any known surgical treatment and where the prospects are as bad as in similar infections of the peritoneum. Case III. is one of these streptococcal cases associated with widespread thrombosis of the cranial sinuses. Fortunately all forms of septic meningitis are not as terrible as these, and quite a long series of less dangerous organisms has already been isolated in many cases. In those here reported they were: Case I., *Micrococcus catarrhalis;* Case II., Pseudo-diphtheria rods, staphylococcus and other bacteria; Case III., streptococcus. But we need hardly be reminded that among others the pneumococcus has been found, also the *Micrococcus fæcalis*, and more whose toxins appear to be far less virulent than those of the streptococcus. Lumbar tapping has apparently been so far useless except for diagnostic purposes in tubercular meningitis.

As to the procedure itself, it is, of course, very simple to anyone who keeps the anatomical details of the lumbar spine carefully in his mind's eye. With a suitable very sharp sterilized hollow needle thrust exactly in the middle line between the second and third, or third and fourth lumbar spinous processes, and consequently below the point at which the cord terminates, it is quite easy to enter the lumbar sac, especially if it is tensely distended with fluid, as is usually the case in meningitis. On the thirty odd occasions on which these three cases were tapped there was not the slightest difficulty in any, although the patients were lying on the side, in which position it is perhaps a little more difficult to keep exactly in the middle line of the body than when the patient is vertical, and where, moreover, the tension of the dura is not so great as in the erect position. The back having been washed with soap and hot water, and subsequently with spirit, and the needle (kept for this purpose and for it alone) having been taken fresh from the sterilizer, there is practically no risk of introducing anything septic into the sac, canal or tissues covering the spine. But here arises a very important question, and I confess I was anxious about it on the first occasion on which I tapped off a greenish purulent fluid from the lumbar sac in Case I. The question is whether in withdrawing the hollow needle the small quantity of septic cerebrospinal fluid which is almost certain to follow it, especially as it is under pressure in these cases, will infect the spinal canal and soft tissues and produce a track of suppuration and possibly a leaking fistula. This fear was soon set at rest by Case I. In spite of every care in withdrawing the needle it was clear that a few drops of fluid did escape on several occasions and trickled for some moments from the puncture. And yet no irritation followed in all his fourteen punctures. Also in Case II., so great was the tension within the dura that a jet 2 in. long followed the withdrawal of the needle after the first puncture and could only be arrested by pressure with the finger. But in none of his fifteen tappings did any irritation of the puncture follow. This was the more remarkable as in one or two of the punctures when the escape was stopped by the finger a visible subcutaneous extravasation of the turbid cerebrospinal fluid of the size of a shilling took place. Yet there was never any sign that this extravasation of septic fluid produced irritation. And in Case III., although the infected fluid contained streptococci, the same was the case. This tallies with the experience of Radmann. He, having had a great difficulty in procuring by cultivation an antitoxin of the meningococcus of Weichselbaum for injection in cases of epidemic cerebrospinal meningitis, conceived the idea of withdrawing the fluid from the dural sac and injecting it as a ready-made vaccine into the areolar tissue of the patient's arm. This he did in two cases to the extent of 8 c.c., and though the fluid was distinctly purulent no irritative reaction was observed in the arm. Both these patients recovered from the meningitis.

Case I.

C. W., aged 31, admitted into University College Hospital on October 17, 1907. The patient seemed an intelligent man, his mental condition was perfectly clear, and his replies to questions quick and sensible. He gave a history of long standing suppuration from the right ear and the opening of an abscess in the ear some years ago. The discharge appears to have ceased a fortnight ago, and since then he has felt cold fits and shivering with bad frontal headache. He had vomited on several occasions without any relation to diet, feeling giddy at the same time. There were no distinctive symptoms referable to the nervous system. No optic neuritis. The pulse varied from 60 to 76; respiration, 16 to 20; temperature, 97·2° F. to 98° F. The right ear showed long-standing disease with loss of the membrana tympani and ossicles, but very little pus or active disease. There was no trace of swelling or tenderness over the right mastoid or temporal region, but there was a slightly enlarged gland below the lobe of the ear. The left ear was normal. In short, there was very little objectively to go upon. Nothing was done on admission but to cleanse the right ear of the small amount of half-dried pus in it. The next day the patient seemed better and was sitting up complaining of very little. Pulse and temperature still subnormal.

About 7 p.m. on October 19 he was said to have had a kind of fit. He felt faint and giddy; closed his eyelids or rolled his eyes, and vomited soon after; no loss of consciousness, muscular spasm or rigidity; both knee-jerks +. October 20: as before; deep pressure on calf produced extension of big toe on left, flexion on right; bowels confined; pulse and temperature, as before, subnormal; speech perhaps a little confused, but answered intelligently. As there was some obscurity about the symptoms I thought it better to do the complete mastoid operation at 8 p.m. Only a little half-dried pus was found in the antrum. The dura was exposed above the roof of the tympanum over an area the size of a sixpence, and the temporo-sphenoidal lobe was explored with a fine trocar in three directions. Nothing was found, nor was there any intracranial tension; there was no subdural pus. The wound was drained with iodoform gauze; lumbar puncture was then done to the extent of about 10 c.c. The fluid appeared to the eye perfectly normal, and, on examination subsequently in the pathological laboratory after centrifugation, it was found to be so, except for a few diplococci, possibly from accidental contamination. This point deserves to be specially kept in mind. October 21: condition generally satisfactory; temperature a shade higher. October 22: not so well; complains of headache on vertex; takes food badly. October 23: intense headache has returned, cries out at times with the pain; much more irritable; temperature and pulse rising; absolutely refused ophthalmoscopic examination. October 24: eyes examined by Mr. Percy Flemming; *no optic neuritis;* headache still bad; discharge at dressing of mastoid wound very offensive, mostly from auditory meatus; twitch of right arm and leg (? cause); wanders occasionally; bowels very obstinate. October 25: slight paresis of right external rectus; very irritable, almost violent when wound was dressed; the slightest movement appears to cause increase of headache; other observations impossible owing to irritability; morphia given with good effect. October 27 (5 a.m.): very noisy and violent, for which hyoscin was given; refuses food; chloroform at noon; catheter passed; right arm again twitched during anæsthesia; 10 p.m.: quieter, free from pain in head; complains of pain in back and abdomen (? saline aperient); wandering and talks much nonsense; feeding per nasal tube was necessary. October 28: quieter night without opiates; headache beginning again, and pain but no tenderness of back; slight weakness of right external rectus; pupils unequal but react well to light; far less irritable later, but required hyoscin at about 12 o'clock.

Five days later, November 2: growing much worse; considerable headache at times, during which he absolutely loses control of himself and becomes extremely irritable; cries out and wanders continuously; tested as to taste, the sense seemed normal; no paresis or paralysis, and no marked wasting; reflexes as before; Babinski's sign sometimes obtained in left foot; pupils sometimes unequal, but react well. Pain and much tenderness over right side of head; no signs of thrombosis; discharge foul as before; temperature irregular, rising once to 101° F., then went down to below normal. On October 31 two rigors without increase of fever; pulse, high tension but slow compared with temperature; *no optic neuritis;* when out of pain very reasonable and intelligent. As the drainage was not good I reopened the wound under chloroform on November 3 and washed out the mastoid cavity. It was then seen that there was much intracranial tension, forcing a little cerebral tissue through the previous puncture in the dura. The bony opening in the skull was slightly enlarged; no subdural pus found. The under aspect of the temporo-sphenoidal convolutions was then explored with a fine trocar, and a spurt of turbid fluid came away under great pressure. Patient breathed more freely after this, and pulse tension was much improved. About 30 c.c. or ℥j. of this turbid fluid were caught in a sterilised test tube, besides which a good deal more was lost. It was not offensive and of light brownish colour. A metal drainage-tube was inserted obliquely forward, apparently between the convolution and dura. When the fluid escaped, the tension on the exposed part of the convolution was relieved. Dressed with iodoform gauze. A lumbar puncture was then made, and 10 c.c. of *thick, turbid, greenish yellow* fluid withdrawn. No definite nervous symptoms on recovery from chloroform, but began to complain of his back. After operation very restless night; had some relief from morphia. Blood-pressure, 100; pulse 108. A good deal of discharge in dressings. November 5: very violent last night and had to be removed to separate room with male keeper. Much discharge on being redressed, for which chloroform was necessary; lumbar puncture as before; 20 c.c. of fluid removed, still greenish, but less turbid. Dr. Thiele reports that the fluid withdrawn on November 3 contained innumerable diplococci resembling *Micrococcus catarrhalis*. They are, like the intracellularis of Weichselbaum, easily cultured and negative in the Gram staining. Further observations set on foot. Patient apparently much better after this lumbar puncture and did not require to be removed from the ward for the night. Facial aspect quite good and patient quite rational; no optic neuritis; takes food well

November 7: excitement, headache and delirium returning; redressed under chloroform: lumbar puncture; 20 c.c. fluid removed; about same appearance, but paler perhaps. Next day, November 8: brighter and better; takes food well; quite rational most of day and not so noisy. November 9: redressed under chloroform; wound better; lumbar puncture, 20 c.c., clearer and less green. November 11: Much quieter on the whole, though drowsy and restless at times; never difficult to manage now, nor is he delirious; has not been in a separate ward for some time. November 12: still much offensive pus from mastoid wound; lumbar puncture, 20 c.c., fluid clearer than last. Pathologist reports that the smaller lymphocytes are on the increase relatively to polymorphonuclear. November 13: quite rational, but a little heavy; signs as before. November 16: lumbar puncture, fluid still clearer; much discharge from cranial wound, especially on introducing a sinus forceps and tilting them upwards; wound of mastoid very offensive; bowels very constipated, enema causes much pain: mental condition good; a little noisy at night sometimes. November 17: redressed without chloroform; on probing mastoid wound patient complained of a burning pain in the tongue tip; syringing the wound caused much pain, which almost made patient lose control of himself. Later in the day patient was a little delirious; had also, about 2 p.m., a slight shivering fit, and complained of feeling suffocated; this feeling lasted only a few minutes; complains of offensive fluid passing from the ear to the throat and producing a nasty taste. November 19: Not quite so well the last few days; delirious at times, at other times quite rational; appetite improving but capricious; headache at times and nasty taste in mouth as before; lumbago (? soreness from punctures); the latest lumbar puncture shows the neutrophiles to preponderate; smoked a cigarette last night; wound still discharging offensive pus, but less in amount; larger tube for drainage goes in 1½ in. to 2 in. into the cranial cavity forwards and downwards; lumbar puncture again. November 23: lumbar puncture, 20 c.c. fluid, a little turbid still. November 24: boric fomentations now; general conditions good; is "silly" at times; drainage-tube had to be shortened to-day; less discharge. November 25: improvement continues; boric fomentations as before; temperature a little raised; sometimes headache; sleeps fairly well; "lumbago" less. November 26: drainage-tube enters with difficulty. November 29: tube omitted; fomentations continued; sits up reading the paper; quite intelligent and rational; conversation at times erratic, especially at night, otherwise sensible enough; bowels more regular than before; still a little headache at times.

December 1: quite sensible now; mental wandering not so frequent; enjoys food; reads paper; lower limbs much wasted; pupils equal, react normally; very deaf in left ear; cranial nerves normal: knee-jerks not so brisk as before but equal; no paresis of limbs; no sensory changes; temperature steady last three days. December 3: general condition satisfactory; lumbar puncture after sitting up for an hour, 20 c.c. withdrawn: fluid appears quite normal to eye (*vide* Table). December 5: doing well; lumbar puncture, 10 c.c., quite clear; no organisms on stain or culture. December 6: condition excellent; now only a button of granulation over cranial wound, practically no discharge. To get up to-day and sit by fire. December 7: improving still in general condition; lumbar puncture, 10 c.c. withdrawn; no organisms on staining or culture; temperature under normal for several days; gaining strength steadily; pulse 80 to 100. December 16: getting on well in every way; weight increasing.

January 4: now looks fat and strong and cheerful; going out in couple of days. January 10: exhibited at Clinical Section, Royal Society of Medicine, looking the picture of health and with no abnormal nervous phenomena.

Case I.—The Lumbar Punctures were done on the following Days:—

October 20 (third day after admission—day of first operation).—First lumbar puncture; proved to be normal in every respect, except a few diplococci.

November 3.—Second lumbar puncture; 10 c.c. thick turbid greenish yellow fluid; deposits largely in test tube; was found by pathologist to contain innumerable *Micrococci catarrhalis.*

November 5.—Third lumbar puncture; 20 c.c., lighter greenish colour than before, but very turbid.

November 7.—Fourth lumbar puncture; same amount and appearances.

November 9.—Fifth lumbar puncture; 20 c.c., fluid clearer and paler.

November 12.—Sixth lumbar puncture; 20 c.c., still clearer and losing yellow colour.

November 16.—Seventh lumbar puncture; 20 c.c., still better in all respects.

November 19.—Eighth lumbar puncture; neutrophiles preponderate.

November 23.—Ninth lumbar puncture; 20 c.c., fluid still slightly turbid but pale.

December 3.—Tenth lumbar puncture; 20 c.c., fluid appears quite normal to eye.

December 5.—Eleventh lumbar puncture; 10 c.c., quite normal to eye, sterile on staining and culture.

December 7.—Twelfth lumbar puncture; 10 c.c., quite normal, sterile on staining and culture.

December 16.—Thirteenth lumbar puncture; 10 c.c., quite normal to eye, sterile on staining and cultures.

January 4.—Fourteenth lumbar puncture; 10 c.c., quite normal to eye, sterile on staining and culture.

CASE I.—C. W.

Date	Number of cells per cubic millimetre	DIFFERENTIAL COUNT		Character of fluid to the eye	Presence or not of bacteria
		Polymorphonuclear cells	Other lymphocytes		
Oct. 20	—	—	—	Normal	Few diplococci (? contamination)
Nov. 8	?	95 per cent.	5 per cent.	Thick greenish	*Micrococcus catarrhalis* countless
,, 11	6,600	29 ,,	70 ,,	Turbid greenish	*Micrococcus catarrhalis*, not so many
,, 18	10,000	58·8 ,,	41·2 ,,	Less turbid	*Micrococcus catarrhalis*, a few still
,, 27	Few	Few	Few	Still less turbid	Ditto and some Gram staining diplococci and rods
Dec. 3	150	,,	,,	Nearly clear	A few *Micrococcus catarrhalis*; no others
,, 5	300	Very few	—	Clear	None on culture or by staining films
,, 7	120	—	—	,,	None after four days on culture
,, 16[1]	2,000	90 per cent.	10 per cent.	,,	None seen or on cultures
Jan. 4	150	20 ,,	80 ,,	,,	None

[1] This count was repeated several times, as there was such an increase of total leucocytes as to suggest an error. The several counts proved the estimate to be correct.

CASE II.

S. C. B., aged 17, a delicate-looking lad, admitted into University College Hospital on December 2, 1907, for "trouble in his head" of two months standing. He had had disease of both ears for two years. On the left this is quiescent now, though there is moisture still present. On the right some discharge can be seen coming through a small perforation in the membrana tympani, which is inflamed. There is no trace of swelling or tenderness over the mastoid or side of the head. During the last two weeks he had suffered from notable headache. On November 30 there had been a "fainting attack" and he vomited soon after. For the last two days before admission the headache had been more intense and his neck had become stiff; the bowels were regular, and he was becoming drowsy. There also appeared to be some slight weakness on the left side, but this was doubtful. There were nystagmoid jerks on looking to the right (confirmed by Mr. P. Flemming). There appeared to be slight weakness of the left rectus; reflexes inconclusive; there was some optic neuritis, more marked in the right eye. The pulse was 55 to 60, irregular; temperature, 99·6° F. in the rectum. Some tenderness on the right side of the neck. The sickness which followed the fainting fit on November 30 has not been repeated since. In bed he lay curled up on right side, and was apparently deaf or drowsy; when roused he replied intelligently. He was not unduly wasted. Any signs present were so indefinite that on seeing the lad I decided to do nothing immediately beyond the cleansing of the ear.

December 3: condition as before, except that the optic neuritis was more marked on the right; abdominal reflexes distinctly diminished on left. On the same day, at 2 p.m., I punctured the lumbar dural sac and drew off 20 c.c. of very turbid fluid; on withdrawing the needle a spurt 2 in. high of cerebrospinal fluid followed, indicating the great tension within. The boy was under chloroform on his side. The fluid, examined by Dr. Thiele, showed swarms of rod-shaped organisms (*vide* full report) and a large excess of polymorphonuclear cells; total lymphocytes, 3,600 per cubic millimetre. At 10 p.m. the temperature was 101° F.; the pulse slow as before and irregular; examination of chest and abdomen negative. December 4 (10 a.m.): I made a second lumbar puncture of 20 c.c.; the fluid was turbid as before; the patient had been semi-recumbent for about an hour previously. The resident medical officer had just before this examined the lad and found undoubted paresis of left arm and leg, but no real facial paralysis. The temperature for the day was variable, as also the pulse from time to time, though usually slow. Still lies curled up in bed on one side or the other; neck still stiff but not so tender in neck muscles as before; had severe headache in the early morning; sleeps badly; eats well; bowels regular. Mr. P. Flemming's examination of eyes showed optic neuritis + 2½ D. on right, 1½ D. on left; paresis of left external rectus; nystagmoid jerks to right; paresis of the associated movements to the right. December 5: fair night; complains of headache when propped up in bed; nervous phenomena not so marked as yesterday; third lumbar puncture, fluid 20 c.c., of same physical characters, drawn off with less pressure. December 6: fourth lumbar puncture, 20 c.c., fluid still as before; lad distinctly better; all nervous signs less marked. December 7: fifth lumbar puncture, as before, 20 c.c. of same quality of fluid; Dr. Thiele reports no organisms in fluid drawn off yesterday; condition of patient as before, but headache clearly increased by propping up in bed. December 8: patient was noisy last night, but became quiet on the injection of $\frac{1}{7}$ gr. of morphia, and remained so during the day; more drowsy and head more retracted; slight facial paresis was also seen; knee-jerks more brisk on left than right; otherwise condition unchanged. December 9: much quieter last twenty-four hours and slept well; left knee-jerks decidedly more brisk;

other nervous signs as before; sixth lumbar puncture, 20 c.c., the fluid was more turbid than before, and had now a yellowish tint. December 10: not so well this morning; slept well first part of night, badly later; temperature, 100·8° F.; he vomited early in the morning for the first time since admission; lies curled up in bed and is irritable: pulse, 64; much tenderness in neck and more head retraction. Seventh lumbar puncture, 20 c.c., the fluid is more turbid than on last occasion, and distinctly more yellowish green; while the fluid was being withdrawn the respirations decreased markedly in amplitude but soon recovered, and the pulse was 56; wasting for the last few days has been rapid. December 11: condition much the same, but the retraction of the head is still more marked and may be said to be extreme; pulse, 60; temperature, 100° F., varied during the day; took food badly, and cried out with headache; Dr. Thiele reports that there has been the greatest difficulty in obtaining cultures from the fluid drawn off on many of the usual media, but that at last some of them are showing signs of growth; what had been withdrawn on December 7 was inoculated at once on sterile cerebrospinal fluid and several other media by Colonel C. Birt, R.A.M.C., who was present, and on other media, but up to December 12 nothing had grown.

As the temporary, though decided, improvement following on the first four punctures of the dural sac up to December 7 had not been maintained, and the patient was becoming more drowsy, with increased optic neuritis, wasting, and head retraction, I thought the time was coming to explore the brain, especially as the left side of the face was becoming weaker and weaker. There had at no time been any fits of any kind, and except the facial weakness and some occasionally in the left arm and leg, nothing distinctive to go upon as to localisation.

December 12 (2 p.m.): I trephined 1 in. above and behind the meatus. There was no subdural pus and no excess of meningitic fluid over the convolutions; the brain surface exposed was congested and bulged; on puncturing it turbid fluid escaped and was caught in sterile test tubes; it was thick and flocculent; the puncture was dilated and a large abscess opened, into which a thick pewter drainage-tube was inserted for about 1¾ in., when pus to the extent of quite 2 oz. escaped into test tubes; the wound was washed and dressed with gauze after partial closure. The lad bore the operation well and spoke quite rationally during the dressing; his colour improved most markedly before he was put back in bed and became a natural rosy tint; his pulse also rose and became more normally rapid and stronger. The first dressing was soaked through in a couple of hours and was changed, which he bore well, and while doing this I noticed that the head retraction had much decreased already and the movements of the head were freer and not so painful. December 13: restless first part of night, better afterwards; temperature rose to 100° F.; at morning visit he looked and said "was a lot better"; less head retraction; lies on left side; takes fluid food very well now; pulse good, 72; temperature 98·4° F.; facial paresis on left almost gone. December 14: better night, but a little drowsy this morning; head retraction almost gone; face better; eighth lumbar puncture, 20 c.c., fluid much less turbid and almost free of colour. December 17: improving, but face on left still weak; head retraction gone; temperature last night rose to 101° F.; pulse at visit, 92; temperature then normal; much red serous discharge from brain; takes food well; bowels acting. For the next three days condition much the same, but considerable headache and slight facial paresis remain; discharge free. December 21: seems considerably better and takes food well; is also brighter, but much wasted; fomentations applied to wound over silver drainage-tube; eyes (examined by Mr. Percy Flemming) showed much less optic neuritis = + 1·5 to 2·0 right and + 0·5 to 1·0 left. December 22: does not seem so well to-day, though he ate a good breakfast; he wandered during the night and pulled off the dressings and drainage-tube, and the fever had risen irregularly as well as the pulse; the wasting continues marked in spite of improved appetite; at 5.30 I punctured the lumbar sac and drew off 20 c.c. of clear fluid under great pressure; still slight facial weakness; no nervous signs in the limbs. As I was leaving town on this day for Christmas I overhauled the patient with my colleague, Mr. Trotter, who was to look after my wards, and put him in possession of all the facts of the case. I expressed the opinion that the boy was improving, but that there was possibly some deficiency of drainage from the brain, or possibly a second focus of suppuration which was still keeping him back, and asked him to keep a close watch on the case and act as he thought necessary. On Christmas Day Mr. Trotter made another lumbar puncture, as I had suggested, and the 20 c.c. of fluid was again slightly yellow; this relieved the headache and the patient had a good dinner, but at 7 p.m. he complained of headache again; his memory was also affected; fearing retention in the original abscess Mr. Trotter enlarged my trephine opening and explored the brain, but no pus was found. December 27: intense frontal headache still and loss of memory. December 28: condition about the same; temperature normal since operation on December 26; although there were no external signs of mastoid disease Mr. Trotter thought it better to open the mastoid cells, and on doing so found them full of granulations and pus under pressure;

the wall of the sulcus lateralis was softened and destroyed; there seemed to be no increase of pressure in the cerebellum, but there was pus in the sulcus over the lateral sinus. December 30: after this operation the patient had less pain; his appetite continued good; temperature still down to normal, but the facial paresis was more marked since the operation on December 28; there was also a good deal of tenderness over the jugular vein, and the right eyelid was œdematous; muscles of two limbs equal; fearing some mischief in the lateral sinus Mr. Trotter tied the deep jugular vein in the neck and opened the lateral sinus; both were normal to the eye; at the same time the cerebellum was punctured but no pus was found; the clearing out of the mastoid cells seemed to have improved matters considerably.

January 1: appetite good; headache less; memory distinctly better; temperature still normal; still some facial weakness on left; from this on to January 14 the improvement was steady in all respects. January 4: Mr. Flemming's examination of the eyes shows great improvement: right less than +1·0 D.; left 0·0 D. On my return to town I made a lumbar puncture and found cerebrospinal fluid normal to the eye; the number of cells in it now was only 300 per cubic millimetre, and there were no bacteria to be seen; from this on to January 15 there was marked improvement of the general condition and of all the special symptoms, and the optic neuritis had quite disappeared on the left and was only very trifling on the right. On January 15 he was obviously not so well; temperature 101·4° F. He had vomited and had much frontal headache on the right side. Lumbar puncture brought away 15 c.c. of clear fluid under moderate pressure. Fearing retention of pus somewhere I exposed the mastoid wound and searched, but found none; the lateral sinus was also punctured but was free of clot; the cerebellum was also explored through the former opening but with negative result. I also shaved off the small temporal hernia cerebri and again searched the brain for pus, which was not found. From this on to the end of January he steadily improved in every way and gained weight and high spirits. Then during the first week of February he fell off again, vomited occasionally and lost his spirits. As I had to leave England professionally on February 5 for some days I again asked Mr. Trotter to watch him particularly. Returning on February 10 I found the boy with a good deal of headache and bulging of the hernia cerebri. On February 11 he was very drowsy and vomited independently of food; temperature normal; left hand growing weak; left knee-jerk more marked than right and facial paresis on left returning.

Having caught influenza myself, I was now obliged to give up my wards for a fortnight and asked Mr. Trotter to watch the boy closely, as I suspected a return of the temporo-sphenoidal abscess. All the symptoms became more marked now daily, especially drowsiness, slowness of pulse and optic neuritis with great increase of the hernia cerebri. The left upper and lower limb on February 15 showed marked hemiparesis and ataxy with incontinence of urine and fæces. Lumbar puncture was done on this day by Mr. Trotter and 10 c.c. of perfectly clear fluid was drawn off. As it was Saturday afternoon the bacteriological department was closed and no detailed observation could be made of the condition of the fluid. Mr. Trotter then cut away part of the

CASE II.—S.C.B.

Date	Number of cells per cubic millimetre	DIFFERENTIAL COUNT			Character of fluid to the naked eye	Presence or not of bacteria
		Polymorphonuclear	Lymphocytes	Endothelial cells		
1907						
Dec. 3	3,670	72·8 per cent.	20 per cent.	7·2 per cent.	Slightly turbid	Rods with polar staining
,, 4	2,100	76·2 ,,	19·6 ,,	4·2 ,,	Ditto	None seen on any cultures, whether blood-serum, glucose agar, gelatine milk, broth
,, 5[1]						
,, 6	1,000	75·8 ,,	20·4 ,,	3·8 ,,	Less turbid	None seen; no growth or cultures
,, 7	18,000	74·1 ,,	22·7 ,,	3·2 ,,	Turbid	Ditto
,, 9	14,000	75·8 ,,	22·4 ,,	1·8 ,,	More turbid	Ditto
,, 10	16,000	78·8 ,,	19·0 ,,	2·2 ,,	,,	Ditto
,, 14	3,000	70·0 ,,	30·0 ,,	—	Less turbid	Ditto
,, 19	19,000	58·0 ,,	40·0 ,,	2·2 per cent.	Slightly turbid	Ditto
,, 22[1]						
,, 25[1]						
1908						
Jan. 4	300	42·8 ,,	56·0 ,,	1·2 ,,	Clear	Ditto
,, 15[1]	—	—	—	—	,,	
Feb. 15[1]	—	—	—	—	Perfectly clear	

[1] Not examined.

Cytolysis was, on December 4, not marked; and on December 19, marked.

The results of cultures from the first tappings in this case were found by Dr. Thiele to be difficult to differentiate. They were ultimately found to be: (1) A Gram staining short pseudo-diphtheria rod; (2) Staphylococci; (3) A non-Gram staining rod, non-motile, coagulating milk and fermenting saccharose; (4) An anaerobic rod which died out.

hernia cerebri and found a large abscess had reformed in the temporo-sphenoidal lobe under the hernia. From this 5 oz. or 6 oz. of pus were evacuated and a metal drain was left in. The next day the drowsiness continued, but on February 17 the boy had much improved, though the hemiparesis continued. From this on there was steady improvement, though on February 20 Mr. Flemming reported that the optic neuritis was worse than at any of his previous examinations, being +4 D. on right and about +2 D. on left. On February 24 the note is of much improvement; goes out on the balcony three or four hours daily. Very little hemiparesis left now and Mr. P. Flemming reports much less optic neuritis.

On my return to my wards on March 2 I found the boy immensely improved. He was cheerful and intelligent and had put on much flesh. In the situation of the hernia cerebri there was still a raw surface, but it was healing to all appearance in a healthy manner.

Case III.

Lilian S., aged 16, admitted on February 2, 1908, with a history of drowsiness, headache and daily vomiting since January 28. She had also suffered from earache for two or three weeks and some stiffness in the muscles of the neck. The trouble in the right ear dates from measles when aged 7, since which there has been discharge on and off. On admission the temperature was 103·4° F.; pulse 78. There was no optic neuritis, and there were no definite nerve symptoms: drowsiness and apathy were well marked. The right ear discharged foul pus, but there were absolutely no external signs of mischief in the mastoid region, which looked and felt perfectly normal to the touch. Finding nothing definite to go upon, nothing was done but to cleanse the ear and remove 20 c.c. of fluid from the lumbar sac. It was very turbid and indicated much intradural pressure. The night following was bad as regards sleep, and the fever rose to 104·8° F. At 6 a.m. the patient had a rigor for thirty minutes with temperature 106·8° F.

At 10 a.m. on February 3 I did the complete mastoid operation and found extensive caries extending into the sulcus lateralis, which laid bare the lateral sinus widely. The latter still pulsated. All the pus and diseased bone were cleared away. The removal of carious bone extended over the dura of the temporo-sphenoidal lobe, but, as there appeared to be no tension under it, it was not punctured, The whole mastoid cavity was drained with iodoform gauze. On the next day, February 4, the patient seemed a little better; temperature 98·4° F., pulse 68; but still drowsy. On February 5 I did lumbar puncture again, and drew off 20 c.c. of clear fluid. The wound was dressed. February 6 (8 p.m.): much the same, but temperature 102·2° F. February 8: last night had a rigor, temperature 103·8° F., and this morning another at 10 a.m., and again at 9.30 p.m.; temperature 106° F. But nevertheless she felt well, and said she was better. There was tenderness on the right side of the neck and hardness over the jugular vein. No optic neuritis; no definite abnormality of cranial nerves.

On February 9, at 2.15 p.m., as I was abroad professionally, Mr. Trotter tied the jugular vein in the neck. It was not thrombosed. He then made a wider clearance of bone round the lateral sulcus and opened the sinus, finding it full of foul thrombus. This was not disturbed, but was left to break down into a gauze dressing. The next two days there seemed to be some improvement, though temperature remained high; patient took her meals fairly well; she had two rigors; temperature rose to 106° F. On February 13 she seemed better. During the night there had been another rigor; temperature 105·4° F. February 15: Mr. Trotter exposed the lateral sinus more freely, also the dura over the cerebellum, but found no pus anywhere. Before next morning there were two rigors; temperature 104·8° F. February 16: drowsiness greater, not looking so well. February 17: no rigors, but obviously worse; optic discs normal; the lumbar sac was again tapped of 20 c.c. of foul purulent pus. February 19: patient comatose. Lumbar puncture was again done, and 20 c.c. of very offensive purulent fluid removed, but no relief. Cheyne-Stokes breathing soon followed, and death at 9 p.m.

Case III.—L. S.

Date of puncture	Number of cells per cubic millimetre	Differential Count		Character of fluid to the eye	Presence or not of bacteria
		Polymorphonuclear cells	Other lymphoctes		
Feb. 3	6,500	91·8 per cent.	8·2 per cent.	Very turbid	Films and cultures show almost pure streptococci
,, 5	1,000	82·1 ,,	17·9 ,,	Clear	Same as last tapping—streptococci
,, 17	not	counted	not counted	Purulent, foul	Not considered worth counting
,, 19	,,	,,	,,	Very foul pus	Ditto

The epitome of Dr. Thiele's notes of the post-mortem are as follow: "No abscess of the brain; meningitis, both cranial and spinal; the cranial was almost entirely limited to the base of the brain, and was very marked there. On the cerebral hemispheres there were only a few flakes of lymph. There was also pus in the ventricles. The right lateral sinus, inferior petrosal and cavernous sinuses were full of pus, and there was some pus in the circular sinus. Left sinuses normal. The right middle ear was quite carious and the labyrinth full of pus. The organs were sound, no pyæmic abscesses.

REMARKS.

These three cases appear to illustrate forms of meningitis less common as to their mode of infection than usual. There appears to have been in each a subacute infection grafted on a chronic, without visibly aggravating the original condition. Thus there were not in either case the slightest external signs of any mastoid or acute ear trouble from beginning to end. The ear, however, was obviously the vulnerable point through which the infection took place, but the way in which the symptoms came on and the whole sequence of events is not usual. In Case I. the collection of inflammatory fluid evacuated clearly lay in the temporal fossa, between the convolutions and the dura, and spread from thence down the spinal canal. In the second case an abscess lay in the substance of the convolutions and appeared to come from a more or less diffuse meningo-encephalitis. Pathologically the two cases were much alike, except in the organism found, but anatomically and clinically they differed. Among the differences clinically are to be specially noted the absence of optic neuritis and definite nerve symptoms in Cases I. and III., while both were well marked in Case II. In Case III. there were also no external signs of the extensive bone mischief present in the mastoid process, and in it, as in the others, the lumbar puncture gave the most valuable evidence of the condition of things within the cranium.

In reflecting on these and allied cases one is brought face to face with facts of great interest. First we see that a patient may suffer from severe non-epidemic meningitis in which a septic semi-purulent fluid, swarming with bacteria, is evacuated from both the cranium and lumbar dural sac, a condition associated with fever, rigor, violent mania or torpor for two weeks, and with great wasting, and yet who is capable of complete recovery on repeated evacuation of the inflammatory septic fluid from the dural sac of the cord without any introduction into it of antiseptics. Moreover, it was found that after a few tappings, and while the cranial wound was still discharging, the fluid taken from the lumbar sac had become sterile as tested on numerous culture media, though still turbid with leucocytes. This would seem to suggest the possible presence of powerful antibodies in the cerebrospinal fluid hostile to the propagation of bacteria. This view gains support from the fact that all attempts to cultivate the organisms in these cases on healthy cerebrospinal fluid failed; nevertheless this fluid is said by some pathologists (Mott) to offer a good soil for the culture of the bacteria. Some experts, however, deny the presence of antibodies in the liquor spinalis, while others believe they have proved their presence, at all events for syphilis (Plaut and Wassermann). But if they do exist for one infection it is not too much to suppose that they may also exist for another; at all events the rapid and absolute disappearance of bacteria from the cerebrospinal fluid after a few tappings, and while the canal was still open to the infected cranial cavity and still contained crowds of leucocytes, is a very remarkable fact. Again, it is interesting to note that the presence of lymphocytes was found long after the cerebrospinal fluid had become sterile. Their differential count in each case followed the usual course, I believe. With the increase of the intensity of the inflammatory process the relative number of the polymorphonuclear cells increased as contrasted with the other forms of lymphocytes; with the decline of the septic activity the relative number of polymorphonuclear cells declined, while that of the others rose. The aggregate total of all forms was of course greatest at the height of the inflammation, and rose to 10,000 per cubic millimetre in Case I. and 19,000 in Case II., falling during convalescence to 120 and 300 per cubic millimetre respectively. Widal, in commenting on a case in which he treated epidemic cerebrospinal meningitis by injection of 0·05 of collargol by lumbar puncture, attributed the great relative increase of the polymorphonuclear leucocytes to the stimulus of the injection and the destruction of the bacteria when acted on by the silver compound to these cells.

Another fact about the first two cases is their complete recovery without obvious damage to the nervous structures from so extensive a meningitis. This is a matter of wonder when we consider the delicate structure of the nervous apparatus. Nevertheless, we find its parallel to some extent in the rarity of damage to the nerves of the extremities after extensive cellulitis with sloughing. In the epidemic form of cerebrospinal meningitis it is stated, on the other hand, that many patients who escape with life are ever after left blind, deaf, or mentally defective. But these and other cases of otitic meningitis with complete recovery suggest that this most important part of our bodies is either *not* so very vulnerable to inflammation, as has been hitherto commonly supposed, or is surrounded by vital defensive works which are designed to protect it in a way not fully appreciated until quite recently. We all remember the views held by our forerunners as to the extreme vulnerability of the peritoneum to septic inflammation. Yet most of us now recognise it as one of the structures pre-eminently capable of taking care of itself and that it can under certain circumstances dispose of an enormous amount of septic matter and recover in the end without operation; witness the many cases of septic peritonitis (*e.g.*, following appendicitis) which recover with or without operation. May it not be that the great serous sac which contains the brain and cord has much the same power of defence up to a certain point? In these two cases the fluid evacuated by lumbar puncture was thick, turbid and greenish, especially in the first, and swarmed with bacteria. And yet, when the tension was relieved by lumbar puncture and the toxins were removed to make place for fresh fluid, possibly containing antibodies, improvement took place regularly each time, especially in Case I., where the delirium and violence were immediately relieved and the patient became quiet for twenty-four hours and then for longer and longer periods until the balance of the nervous system was completely restored. Parallel with this went the disappearance of the bacteria until the fluid was found sterile. In a closed cavity the toxins appeared to operate in all their force until relieved by puncture and evacuation; something in the nature of antibodies and the phagocytes appeared to be able to cope with the reduced number of bacteria.

The question whether this systematized tapping of the lumbar sac in the epidemic form of meningitis could with advantage be combined with the injection of antiseptic fluids into the spinal dura has occurred to many physicians, and has been put in practice by some, who have expressed the opinion that they have improved their results thereby. Thus Franca injected 3 c.c. to 9 c.c. of a 1 per cent. solution of lysol in children and 12 c.c. to 18 c.c. of the same in adults, and, he believes, with advantage. Papillon, Laurens, Widal, and Raymond employed collargol, with a high percentage of recoveries. But when one compares these results with those of cases treated by simple repeated tapping without antiseptic injections one carries away the impression that the chief factor in the recovery was the systematised evacuation of the toxins from the canal; again, the disappearance of the bacteria from the cerebrospinal fluid after several tappings, vouched for by thoroughly competent bacteriologists, lends support to this view; moreover, the failure of germicides to arrest septic inflammation in the abdomen seems to contra-indicate their use in the dural canal. If it be true that there exist in the cerebrospinal fluid antibodies hostile to the bacteria, as some believe, either originally present or furnished under the stimulus of inflammation, it may be enough to relieve the dura of its overdose of toxins and to encourage the reproduction of fresh cerebrospinal fluid to assist in the recovery of the patient. At any rate, the results obtained by repeated lumbar tapping in the epidemic cerebrospinal meningitis are encouraging, and one may hope that in some of the non-epidemic forms, such as the cases here recorded, we may have similar encouragement when the procedure has a further trial.

In conclusion, I should like to say that it would have been impossible to make the records of these cases as complete as they can claim to be without the cordial coöperation and unflagging industry of my house surgeons, Messrs. Owen and Appleyard, and of our pathologist, Dr. Thiele. I am also deeply indebted to Col. Birt, R.A.M.C., for several independent bacteriological investigations of the products of our tappings, which, at great trouble to himself, he was good enough to make.

REFERENCES.

SEVESTRE. *La clinique infantile*, 1905, No. 9.—A child, aged 7, tapped twelve times in a month to the gross amount of 350 c.c., with improvement each time and complete recovery.

C. FRANCA used 3 c.c. to 9 c.c. of 1 per cent. solution of lysol in children and 12 c.c. to 18 c.c. in adults. *Deutsch. med. Wochenschr.*, 1905, xxxi., p. 789.

PAPILLON reported on February 6, 1906, to the Société de Pédiatrie de Paris a case in which he had injected a child, aged 3½, with very severe symptoms of cerebrospinal meningitis, with 2 c.c. to 4 c.c. of a 1 per cent. solution of collargol into the subarachnoid (? subdural) space with a good result. He admits, however, that where the exudation on the spinal cord is very thick and prevents the cerebrospinal fluid from circulating there is little hope of recovery.

VORSCHUTZ, *Münch. med. Wochenschr.*, 1907, liv., p. 514, records five cases of acute epidemic cerebrospinal meningitis with Weichselbaum's bacillus treated by lumbar puncture, with complete recovery in four.

LENHARZ, of Hamburg, appears to have been the first to demonstrate the great value of systematic lumbar puncture in these cases. (*Münch. med. Wochenschr.*, 1905. lii., p. 537; *Deutsch. Archiv. f. klin. Med.*, 1905, lxxxiv., p. 81).

BÖKAY records ten cases of recovery from cerebrospinal meningitis in patients, aged from six months to 12 years, treated by lumbar puncture. (*Deutsch. med. Wochenschr.*, 1907, xxxiii., p. 1947.)

Laurens showed a case to the Soc. Méd. d'Hôp., November 15, 1907, of acute general septic meningitis, due to the enterococcus and following ear disease, in which recovery took place after daily lumbar puncture with injections of 5 c.c. of electrargol into the dural sac.

Widal and **Ramond** also reported a similar case with recovery following lumbar puncture and injection into the dural sac of 0·05 c.c. of collargol.

Radmann. *Semaine Méd.*, Par., 1907, xxvii., p. 383; *Centralbl. f. Chir.* 1907, xxxiv., p. 1277.

Lenharz, *Centralbl. f. Chir.*, 1907, xxxiv., p. 1277, has had fifty sporadic cases of epidemic cerebrospinal meningitis with 51 per cent. of recoveries, and in the recent epidemic of the same fifty more with 37 per cent. of recoveries. He speaks highly of systematic spinal puncture.

Wilms, in the same discussion, also speaks highly of puncture.

Müller also praises it and supports Lenharz.

DISCUSSION.

Mr. Charles A. Ballance said he thought all were greatly indebted to Mr. Barker for bringing forward the cases he had and for the admirable résumé of the work which had been done. He did not pretend to have followed the details of his cases, but the remarkable facts which the author had related concerning the withdrawal of the purulent fluid by lumbar puncture must strike all, and those who had practised the methed in meningitis would be encouraged in the future. He (Mr. Ballance) had published some cases of the same sort, and had been much impressed with the great advantages of the method. He thought that cases of meningitis naturally divided themselves into meningitis serosa and meningitis suppurativa. The first of these, certainly in some cases, might be looked upon as a stage previous to meningitis suppurativa: and if by treatment the first kind, or serotoxic meningitis, could be arrested, an immense gain was effected because, while one may hope to deal successfully with meningitis serosa, meningitis suppurativa must often be beyond hope of treatment. The collection of fluid in meningitis serosa must be either in the subdural or the subarachnoid space. Those spaces were quite separate, and it was natural that fluid should collect in those spaces when there was a suppurative area in their vicinity, just as all were familiar with the collections of pus in the pleura or in the peritoneum, with larger collections of serous fluid in their immediate vicinity. In a limb an area of inflammation was first represented by œdema, but in the cavities in the skull not œdema but a pond of fluid formed, and if that pond could be tapped, no doubt in many cases the suppurative stage might be prevented. The symptoms which he had observed in meningitis serosa were a rise of temperature, a slowing of the pulse, nausea and vomiting, sometimes drowsiness and partial stability in the movements of the pupil. Such often arose in patients with very acute otitis or otitis spreading into the mastoid process, and in those in which a mastoid operation was indicated. When those symptoms occurred, he thought lumbar puncture should always be associated with the mastoid operation, and, as Mr. Barker said, the puncture should be repeated if the symptoms returned. He had known several cases in which lumbar puncture had brought away 2 oz. or 3 oz. of fluid under pressure, and all the symptoms had immediately disappeared. With regard to meningitis suppurativa, when there was turbid fluid in the lower part of the lumbar theca, as Mr. Barker's cases had shown, and as he, Mr. Ballance, had also had experience of, it did not necessarily mean that the case would be fatal. Even that turbid fluid would give way to repeated lumbar puncture, and in that way great assistance was given in the management and cure of such cases. He did not think that suppurative meningitis of a fulminating character could be expected to yield to such a method of treatment. But there were milder forms of meningitis; for instance, on doing lumbar puncture in some cases of brain abscess it happened that there was turbid fluid at the lower part of the spinal canal, and before opening the brain abscess it was thought that the patient could not recover. But though the fluid was turbid, yet after opening the brain abscess it became clear again and the patient did recover. All the cases which had been mentioned showed very clearly the great advantage of the method of lumbar puncture, and possibly it might be used with advantage in many cases which even now had not been recognised as suitable for it.

Dr. FREDERICK E. BATTEN said that possibly from the medical aspect one took a different view of the fatality of meningitis from that which Mr. Barker seemed to take. The meningitis to which he would refer was that of children, and was known as "posterior basic," which might be regarded as a form of cerebrospinal meningitis, whether epidemic or not. In that form it was well known that a considerable proportion of recoveries normally took place. Before lumbar puncture was ever done the recoveries amounted to between 30 per cent. and 40 per cent. In regard to some of those it might be objected that they were wrongly diagnosed, but others, he thought, did not admit of any doubt. Since lumbar puncture had been introduced, one had definitely proved cases which were admitted into the ward as being instances of cerebrospinal meningitis. In the first series they were usually allowed to take their course, and the percentage given above recovered. Then there came the time when cases were treated with frequent lumbar puncture, but he did not think the results were any better than they had been under the normal course of the disease. Lately, in cases of epidemic cerebrospinal meningitis, injections had been made into the spinal canal, and it would seem certain, from the observations of Flexner, that by the injection within the spinal theca of antimeningococcic serum, a greater proportion of cases had recovered. This form of treatment, however, had still to be proved beneficial in the "posterior basic" variety of the disease. He thought many cases recovered without leaving any after effects. Complete recovery occurred in a definite proportion of cases where there had been very severe meningitis, as could be proved by lumbar puncture. Mr. Barker had referred to the fact that after the withdrawal of the fluid from the spinal canal which was infected with meningococci, it was injected into the subcutaneous tissues without effect. That the meningococcus had very little pathogenic effect so long as it was injected into subcutaneous tissues was well known, and to produce definite symptoms it had to be injected into certain susceptible parts. In Flexner's experiments on monkeys, he injected what he termed colossal doses frequently to produce cerebrospinal meningitis. With regard to Mr. Barker's first case, there was no evidence of infection of the spinal meninges at the first operation, and the infection must have taken place between October 20 and November 8, when *Micrococcus catarrhalis* was found. He thought it might have been introduced after the meninges had been punctured. He did not think one could identify *Micrococcus catarrhalis* with the form of meningitis known as cerebrospinal. If it was *Micrococcus catarrhalis*, it was one of the less virulent kinds, and tended to get well. With regard to the pus evacuated from the second case, rods with polar staining were obtained which did not grow afterwards, and all the other cultures were negative. Then the abscess was evacuated, and he would have liked to know what organism was obtained from that abscess; it ought to throw some light on the form of meningitis which was present. Mention had been made of the severe effects of meningitis on the nervous system; it was curious, if one examined the nervous tissue in cases of severe pneumococcal and meningococcal meningitis, how very little change might be present in the cells of the cortex or in the cells of the white matter of the spinal cord, so that if the meningitis could be cured one could understand the perfect recovery from the nervous symptoms.

Mr. BARKER, in reply, said Mr. Ballance had referred to the removal of 2 oz. or 3 oz. of fluid from the lumbar sac in such cases of meningitis as those in question, but that seemed a very large quantity—about 60 c.c. to 90 c.c. Possibly it was a slip. All recognized the need for discrimination between the several varieties of meningitis; they were perhaps as different as was pneumonia from other chest affections. There were hopes of recovery in some, but in the virulent form, such as in the last case, where there was severe streptococcal infection, the chance of recovery was very small. With regard to the series of cases of which Dr. Batten spoke—cases of sporadic cerebrospinal meningitis, which went a long way back in time—Dr. Batten admitted that many of them could hardly be due to the *Diplococcus intracellularis*. In examining a series of cases which had been treated, those should be excluded in which an organism had not been definitely found. And he had been greatly struck, in reading the records of work done in England and abroad, to notice that where the symptoms were exceedingly severe in the epidemic form, and the special organism was positively found, they had yielded very definitely to tappings. And his paper suggested that if in that form benefit was produced, surgeons might find the method of use in the diseases coming under their ken. With regard to the material from the abscess in Case II., it was examined by Dr. Thiele and Colonel Birt, but they had great difficulty in separating the organisms. He believed they found the same organisms as in the cerebrospinal fluid. The possibility of contamination, which he recognised from the first, was practically negatived by the method employed. He attached a sterile syringe out of the sterilizer to the needle, and as the fluid flowed off the hydrostatic pressure was enough to send back the plunger. Dr. Batten suggested that infection of Case I. might have occurred after the operation on the meninges. That was possible, but he did not think it occurred, because the patient had many of the symptoms of meningitis for many days before admission, and some diplococci were present in the first tapping. The cases were to him encouraging, and might lead to surgeons employing the needle in the spinal canal for diagnostic purposes, and in some cases great benefit might also be produced.

Surgical Section.

May 12, 1908.

Mr. J. Warrington Haward, President of the Section, in the Chair.

Sciatica and its Surgical Treatment.

By J. Crawford Renton, M.D.

In order that you may follow what I intend to say with reference to sciatica, a single sentence on the anatomy of the nerve seems essential. The great sciatic nerve, the largest nerve in the body, is formed from the principal part of the lumbo-sacral cord and first, second and third sacral trunks, that is, from the sacral plexus. It passes out of the pelvis at the great sacro-sciatic notch below the lower border of the pyriformis muscle, being covered with the gluteus maximus, and passes down the thigh, lying between the ischial tuberosity and the great trochanter, being slightly nearer the former. Further on it is covered by the long head of the biceps. Usually it divides somewhere in the upper two-thirds of the thigh, but occasionally the internal and external popliteal nerves spring directly from the sacral plexus. It supplies the hamstring muscles, and sends a branch to the adductor magnus. Branches pass from the sacral plexus and great sciatic to the posterior aspect of the hip-joint. This latter fact is of importance.

For practical purposes the disease called sciatica may be divided into :—

(1) Neuralgia of the nerve.

(2) Neuritis.

(3) Perineuritis.

(4) A combination of neuritis and perineuritis.

All these different forms may be met with either singly or combined.

Causes of Sciatica.

(1) Pain in the sciatic nerve may be due to pressure on the nerve within the pelvis, *e.g.*, a distended rectum, uterine tumour, pelvic inflammation, malignant disease in the pelvis, sciatic hernia, aneurysm of the internal iliac artery, &c.

(2) Joint lesions, tubercular disease of the hip-joint, rheumatoid arthritis, disease of the sacro-iliac synchondrosis, tubercular disease of the spine, including psoas abscess.

(3) Disease of the cauda equina or of the nerve roots, or a lesion of the cord higher up than the sciatic nerve. This includes locomotor ataxia.

(4) Disease of the femur, exostosis, sarcoma.

(5) Chronic rheumatic thickenings in muscles may give rise to symptoms almost similar to sciatica, which have been described by Professor Stockman, also nodes described by Dr. Garrod and Dr. Hawthorne.

(6) Gout, rheumatism, syphilis, ordinary chills.

(7) Mechanical causes, such as blows, long pressure, &c.

Symptoms.

The chief symptom of sciatica is pain, which is felt passing down along the line of the nerve.

(1) In neuralgia it comes on typically at stated intervals and subsides after varying periods, and the pain is of a dull aching character, with occasionally paroxysms of darting or boring pain. A paroxysm may be brought on by movement of the limb or any local pressure, as from sitting on a hard chair. The patient is therefore careful to sit in such a way as to protect the hip from any pressure, and he may have a certain amount of twisting of the spine in order to add to his comfort. The reflexes are normal.

(2) In distinct neuritis the pain is very severe; and although the recumbent position relieves it to a certain extent, morphine subcutaneously is generally necessary. The pain usually extends from the pelvis down the back of the thigh and calf, and may reach the sole of the foot, the most intensely painful spots being the posterior iliac spine, the sciatic notch, the middle of the thigh, behind the knee, below the head of the fibula, behind the external malleolus, and on the dorsum of

the foot. There is frequently tingling, anæsthesia, and, as a result of the pain, atrophy and muscular weakness. In chronic cases, reaction of degeneration may occur. As the neuritis subsides the patient is able to walk about for a time, varying from half an hour to an hour, without recurrence of pain, and this is gradually increased as the pain subsides. The reflexes may be diminished or lost. There are also areas of impaired sensation which do not exist in neuralgia. Both sciatic neuralgia and neuritis are unilateral. Bilateral pains suggest some central cause or some toxic condition, inducing a peripheral neuritis. The lightning pains of tabes may be unilateral or bilateral. They are commoner in the legs than in the arms, as tabes is a disease which affects the root fibres of the lumbo-sacral region, and, of course, local hyperæsthesia of the skin is frequently associated with them. The pains induced by tumour and spinal caries may give rise to unilateral sciatica to begin with, and cases of this kind have been operated upon; but later, the disease having progressed, the true nature of the condition has shown itself and other treatment has been required. A case such as this will be referred to later. In cases of complete pressure on the nerve by tumour or inflammatory exudates, there may be paralysis and atrophy of the muscles. It is well to keep in mind that cases of functional pain in the sciatic nerve are from time to time met with, but the anæsthesia and hyperæsthesia found in those cases is an aid towards eliminating them from cases of genuine disease.

(3) In perineuritis the symptoms are very similar to neuritis to commence with, but the striking feature in cases of this kind is that when the acute attack subsides, the pain is entirely absent when the patient is at rest; but when any attempt at walking is made, it comes on in from three to ten minutes, and sometimes it is so bad on movement that patients are not free from it unless when lying or standing. One patient will be referred to later who was ill for three years, and was compelled to take his food either in the recumbent or standing position.

(4) Neuritis and perineuritis: Cases of this kind have intense pain, both in bed and on slight exertion. The neuritis gradually subsides in ordinary cases under medical treatment, and in a certain number of cases the perineuritis will also pass off; but it is found that where the pain on movement continues, the perineuritis has left a condition of pathological change which medical treatment is unable to relieve.

DIAGNOSIS.

Enough has been said above to indicate the various symptoms of importance to be attended to. We must carefully examine the points of pain, note the difference between the two limbs with regard to muscular atrophy and the presence of anæsthesia and hyperæsthesia, the character of the pain, as to when it occurs, whether it is present when resting in bed or starts shortly after movement. After the acute symptoms have subsided it is more easy to settle whether we have a neuritis, a perineuritis, or a combination of both.

An examination of the pelvis, the spine and abdomen under chloroform having revealed no hip-joint disease, no sacro-iliac disease, no pelvic lesion, and no tumour of the femur or of the iliac bone, and an X-ray photograph showing nothing abnormal, we must decide whether we have to deal with a neuritis or a perineuritis.

The symptoms above detailed are perfectly clear, and with their presence after the acute stage has subsided, there is no difficulty in diagnosing whether a neuritis or a perineuritis exists. After the acute stage of a neuritis is over the pain gradually subsides and the patient is free of pain, but in a perineuritis with a pathological exudate or adhesions around the nerve the patient is suffering from a much more chronic and persistent form of trouble.

PATHOLOGY.

Sciatica is regarded by Sir William Gowers and Dr. Frederick Taylor as a true neuritis in the majority of cases, as shown by symptoms of anæsthesia and muscular atrophy, and by the fact that post-mortem evidence of neuritis has been found. The neuritis may be—(1) Interstitial, affecting mainly the connective tissue, or (2) parenchymatous, affecting first the nerve fibres themselves, or often (3) a combination of the two. Where the neuritis affects chiefly the perineurium or sheath, the nerve fibres may escape serious damage. If it affects the endoneurium, then the nerve fibres are more liable to be affected. Ultimately a new fibrous tissue may be developed in the interstitial tissue and sheath, and around this a varying amount of pathological exudate or adhesions takes place.

In the parenchymatous form the disease begins in the nerve fibres and may lead to serious results. In cases of persistent pressure in intrapelvic or spinal disease one or both nerves may be so altered that their function may be partially or wholly destroyed.

Men suffer from sciatica much more frequently than women.

SURGICAL TREATMENT.

Various forms of treatment have been employed, and they may be conveniently divided into: (1) Bloodless methods; (2) operations that have been from time to time recommended and carried out.

(1) Bloodless methods include forcible flexion of the hip under an anæsthetic, acupuncture of the nerve with long needles or with a needle connected with a continuous current battery.

(2) Operations: Nausbaum, thirty-four years ago, first recommended nerve stretching by the open method, and this has been largely used both at home and abroad. This has not, however, given satisfactory results in a great many cases. Personally I have been so disappointed with it that, on being consulted with reference to a patient in 1895, whose nerve had been previously stretched with only slight benefit, I advised that the sciatic nerve be thoroughly re-examined. The patient, who was a medical man and who had suffered a very great deal, begged of me to examine also the external and internal popliteal nerves. I exposed the sciatic nerve by the usual vertical incision, and found it surrounded by adhesions extending upwards to the sciatic notch. One adhesion was 1½ in. both in length and breadth, and extended from the nerve to the ischial region. This I divided with scissors, cutting away its attachment to the nerve. The others I removed by stripping the adhesions attached to the nerve from the sciatic notch to the middle of the thigh. I also exposed the external and internal popliteal nerves, but found them normal. The patient made an excellent recovery, was bicycling and doing his work in four weeks, and to-day, thirteen years since the operation, he remains perfectly well. His prompt recovery after this operation, considering that he had visited the spas recommended medically for such cases, and had been unable to do his work for more than a year, led me to look carefully into the question as to what cases would be benefited by this operation of removing adhesions from the nerve. I came to the conclusion that the cases which would be benefited were those presenting the symptoms which I have described under perineuritis, viz., pain entirely absent when the patient is at rest, but commencing three to ten minutes after attempts at walking are made. I also noticed that in such cases, after the acute symptoms subsided, tenderness and pressure along the

line of the nerve were absent. It will be evident to everyone that stretching of the sciatic nerve, even to the extent of raising the patient from the operating table by the nerve, in the case I have above described, would be absolutely useless. I am very careful not to stretch the nerve more than is sufficient to enable complete removal of the adhesions.

In two cases, at the end of two and six years respectively, recurrence took place. A second operation was done, removing new adhesions in both cases, and the patients are now quite well. It is possible for a patient to have two or even three attacks of perineuritis.

During the last thirteen years I have operated on thirty-two cases, and all the cases have benefited, not only at the time of operation but have remained well up to the present. To some of the cases operated on I wish to refer as they present points of special interest and importance.

Mr. A., aged 37, from Newfoundland, had suffered for seven years from severe pain and had tried all forms of treatment. Walking was accomplished with difficulty, requiring the aid of two sticks, and he was a complete invalid. I advised operation and found the sciatic nerve surrounded by adhesions, which I removed. It is twelve years since his operation, and he writes me that he never can be thankful enough that he went to the Old Country and got his nerve attended to.

J. D., male, aged 46, had suffered severe pain for three years and was quite unable to do his work. He was so ill that he was only free from pain when lying or standing, and he took his food in the latter position. I operated in the usual way and found large adhesions, especially one towards the ischium similar to the first case I have referred to. This patient made an excellent recovery, although it was six weeks before he was free from pain. This I have noticed in several other cases, being due, I have no doubt, to the long-continued dragging on the nerve, and possibly partly to the habit of pain which may be induced.

W. R., male, aged 52, had suffered from sciatica for five years. The symptoms in the last two years having become more severe he came home from the West Indies to be under my care. He was so obviously suffering from perineuritis that I advised operation, but I warned him and his relatives that they must not expect the pain to be gone immediately after operation. I exposed the nerve and found it pulled inwards by adhesions, which I removed in the same way as I have above described. He made an excellent recovery, and two years after the operation I had a letter from him telling me that he continued quite well and was doing hard work every day.

J. D., male, aged 36, had suffered from sciatica for two years. He had all the symptoms of perineuritis, for which he had visited the usual spas without benefit. I exposed his nerve and removed very firm adhesions from the sciatic notch to the middle of the thigh. In addition I cut out a rheumatic thickening in the gluteal muscle similar to those described by Professor Stockman. It is two and a half years since the operation and the patient has remained quite well.

The only other case to which I wish to refer is that of a man aged 30, who had all the symptoms of perineuritis without any evidence of a central cause. I exposed his nerve, removing a number of adhesions, and he went home with the pain quite relieved. He returned, however, in six months with evidence of Pott's disease of the spine. He was treated for this, and gradually recovered and had no recurrence of the sciatica. This is one of the exceptional cases where a perineuritis has been set up by a tubercular disease. At the present time Calmette's tuberculin eye test might be helpful in clearing up the diagnosis.

These cases are sufficient in number to illustrate the great importance of examining the sciatic nerve for adhesions, which no doubt are the result of a perineuritis. When a case of sciatica is persistent and is not improved by medical treatment, with a trip to one of the spas, and where there is no reason to suspect a tumour pressing on the nerve, and especially where the patient's symptoms show that he is free from pain when resting, then most certainly the nerve should be examined for adhesions. I am sure that small adhesions frequently disappear, but large ones, similar to those I have described in the cases above detailed, will not disappear. Mere stretching of the nerve is not of any service in cases with large adhesions; they must be removed.

In all cases of perineuritis with large adhesions there must always be a certain amount of neuritis due to dragging on the nerve by the adhesions, but this subsides after the operation.

I can heartily recommend the treatment I am advocating for a careful trial, and the fact that after three, four and seven years of suffering it has been the means of returning the sufferers to comfort in life and ability to do their work is strong evidence in its favour.

DISCUSSION.

The PRESIDENT (Mr. Warrington Haward), in thanking Dr. Renton for his paper, said that any contribution towards the successful treatment of such a painful and persistent disease as sciatica must be a great advantage. He asked whether any other surgeons present had examined a nerve in the way described, and if so, with what result. He asked whether Dr. Renton took any steps towards preventing the reformation of adhesions after they had once been removed. Often, especially in the case of the abdomen, it might be easy to remove adhesions, but difficult to prevent their recurrence. The author had pointed out that there were cases in which pain was not continuous, but was only felt when walking was attempted, and that seemed an interesting point in the diagnosis of some kinds of neuralgia to which allusion had been made. It would be interesting to hear the experience of surgeons on that matter, because it could not be doubted that sciatica had many and varied origins, and treatment must depend on its correct diagnosis. Dr. Renton had pointed out the character of those cases of sciatica to be treated by the division of adhesions.

Mr. SWINFORD EDWARDS asked how the author located the adhesions, and what was the length of his incision in a given case.

Dr. CURTIS WEBB said he was not a surgeon, but he was emboldened to make a remark by a sentence in the paper to the effect that the operative treatment of sciatica was worthy of a trial when medical treatment failed, also spa treatment. He did not wish to go into details, but he was particularly interested in the treatment of that and allied conditions by static electricity. Some time ago he published a paper on four very chronic cases of sciatica, of from ten to five and a half years in duration, which were all cured, and remained so. Though he did not doubt that there were obstinate cases, in which everything failed, static electricity was worth trying. He did not mean the high frequency nor the constant current, but Morton's wave form and sparks. That had completely cured some obstinate cases. He had the case of a house physician, aged 29, in mind. He was attached to one of the teaching schools in London, and developed sciatica. He had treatment at the hospital, his nerve being stretched subcutaneously. His father was a wealthy man, and so the patient was able to spend two and a half years wandering around England and the Continent at various spas. But he got no benefit and was practically crippled when attempting to walk, though this did not cause any actual pain. He was advised to try some of his (Dr. Webb's) "quackery," and that was so successful that in three weeks he went away all right. That was August, 1906. He came for a few more applications in the autumn and was now resident officer at another large hospital, which post he continued to hold. He had now had sixty cases of sciatica under that treatment. Five were complete failures, three being in patients aged over 65, but all the others were either cured or relieved, and that showed that before surgery was resorted to static electricity should be given a trial, even in the most obstinate cases.

Dr. RENTON, in reply, expressed his high appreciation of the remarks which had been made. How to prevent the reformation of adhesions was a very important question, especially as all knew the trouble caused by adhesions in the abdomen. But with regard to the sciatic nerve, if one cut away the thick adhesions and stripped off the smaller ones, no trouble ensued on that account. In a fortnight the wound was healed and the patient was allowed to get up and walk about. That occurred in the chronic cases which had been suffering for years. He did no more than satisfy himself that the nerve was free, from the sciatic notch to the middle of the thigh, the region in which adhesions were most apt to form. His incision was an ordinary one of 3 in. to 4 in. in length, which enabled the finger to be passed along the main line of the nerve as far as the sciatic notch, and downwards a considerable distance. In stout persons it had sometimes been advisable to extend the incision upwards a little. He was much interested in Dr. Webb's remarks relative to static electricity, and agreed that there was a great future for various forms of electricity up to a certain point. He did not operate upon any case unless he was convinced that there were adhesions causing the symptoms, nor until all other means had been given a trial, including electricity and change of air. Small adhesions would often disappear without surgery. He would not think of operating on an acute case, and in the last fourteen years he had operated upon only thirty-two cases, a very small number compared with those which he found yielded to medical means.

The Treatment of Ulcerative Proctitis by Zinc Kataphoresis.

By F. C. Wallis, F.R.C.S., and W. Ironside Bruce, M.D.

Ulcerative proctitis, whether originating in the bowel from an infection spreading from the crypts of Morgagni or whether a sequel to colitis or dysentery, has the same symptoms and a similar pathology, as far as the method of destruction of the mucous membrane is concerned. It is this latter which has constituted the difficulty in treating these cases with any measure of success, and it has only been in those rare instances in which the whole ulcerated area could be removed and healthy mucous membrane brought to the skin margin that any definite or permanent cure has been obtained.

The difficulty of any form of palliative treatment being successful is due to the method by which the ulceration progresses when once it has established itself. This is not by the direct destruction of the mucous membrane, but by infiltration of the submucous tissue, which, becoming œdematous, gradually cuts off the blood-supply to the mucous membrane, which in its turn becomes œdematous and gradually disintegrates. In the meantime the infection slowly forges ahead in the submucous tissue.

The other local symptoms are a sense of heat and swelling in the rectum, with pain and tenesmus and diarrhœa, the bowels acting five or six times during the first part of the day, after which the patient is fairly comfortable until next morning. There is a sanious discharge of a peculiar salmon pink tint, which is always present when the ulceration is in progress. Patients are apt to get febrile attacks and to suffer from synovitis of various joints.

Another form of ulceration, which may be termed catarrhal, is one in which the ulceration is superficial, and when inspected by the sigmoidoscope the mucous membrane presents a curious mottled appearance, the pink colour being interspersed with patches of a wash-leather colour. This condition is seen in what is called mucous colitis, and also is usually present when there is some chronic irritation of the bowel, such as is produced by a polypus, papilloma or villous tumour. When the cause is a simple tumour the removal of it is usually followed by a rapid and complete recovery of the mucous membrane. But when this is not the case this variety responds quickly to the form of treatment under consideration.

Other forms of ulceration to which this treatment is applicable are follicular ulcerative colitis and post-operative ulceration due to sepsis. Of this latter variety there are two forms; the one resembles that already mentioned, which starts as an infection from some pre-existing lesion and has all the symptoms of infective ulcerative proctitis. The other form is somewhat rare, but occurs especially after operations for hæmorrhoids in people whose healing powers are indifferent or in abeyance, so that, instead of the wound healing, a chronic granular surface persists, which steadily refuses to heal and is painful when any examination is made and also when the bowels act. The pain is not lasting and there is no sense of fulness or feeling of tenesmus, such as occurs in the ordinary form of infective ulcerative proctitis, nor is there any excessive discharge nor œdema of the tissues around the anal margin. The main characteristics of this form of ulceration are the pain described and the negative quality it exhibits as to any tendency to heal. It is particularly for the infective and for this form of ulcerative proctitis that I have been endeavouring to find some form of treatment which would give a quicker and more definitely beneficial result than those methods which have been tried hitherto with such little success except in those rare instances already mentioned in which some operative measures enable one to get rid of the whole of the affected tissues.

In the *British Medical Journal* of November 4, 1905, there is an interesting report by Dr. J. Curtis Webb of the treatment of a case of colitis by means of electric enemata. The treatment carried out was as follows: After a preliminary lavage of the bowels $1\frac{1}{2}$ pints of a solution of silver nitrate of the strength of 0·1 per cent. were injected through a rectal tube in which was a copper wire that could be connected with the positive pole of the battery. Large clay electrodes were placed on the back and abdomen and connected with the negative pole. A current of from 15 ma. to 20 ma. was passed for fifteen minutes. The result of this treatment was practically a cure, as all symptoms disappeared and the motions became natural in consistency and frequency. Ten electric enemata were given in all.

In 1906 I saw a patient of Dr. Ernest Baker's, who had colitis and considerable irritation of the bowels, causing them to act several times a day. On examining with the sigmoidoscope the mucous membrane was seen to be of the mottled wash-leather character already mentioned, and in discussing the treatment Dr. Baker mentioned the case of

Dr. Curtis Webb's to me, and said he would like to try the same treatment on this patient, which he did with complete success.

Upon this my colleague, Dr. Ironside Bruce and I had various consultations as to the possible benefit to be derived by this treatment, in those forms of ulceration already mentioned which have hitherto been so intractable, with the result that we have tried it in some of the very worst cases with beneficial effect.

Description of the Treatment by Dr. Ironside Bruce.

The introduction of the zinc ions into the tissues by means of galvanic currents has been exceedingly useful in the hands of Dr. Lewis Jones and others in the treatment of rodent ulcer. In chronic ulceration of the rectal wall the same treatment also gives promising results. Shortly, the principle of the treatment is that zinc sulphate is broken up by the galvanic current, the zinc ions travel towards the negative pole and are thus driven into the tissues surrounding the positive pole. The SO_4 so liberated combines with the metallic zinc of the positive pole to form again zinc sulphate. The method of application is exceedingly simple. The necessary apparatus is as follows: A zinc rod, 6 in. in length, with suitable connection at the end for the purpose of attaching it to the positive pole of a galvanic supply; a large indifferent electrode to connect the negative pole. The zinc rod is covered with four layers of lint, which is saturated with a 4 per cent. solution of zinc sulphate (in distilled water). The negative electrode is soaked in plain water to ensure a good contact.

The patient being suitably placed, lying on the side, with the aid of a little vaseline as a lubricant, the positive pole is introduced in the rectum to a distance well above the ulcerated area. The indifferent electrode is placed over the sacrum or on the abdomen: to this is attached the negative pole of the source of the galvanic current, the positive pole being attached to the zinc rod. It is necessary to have a milliamperemeter in circuit. All connections having been made secure, the circuit is completed and the resistance cut out until the meter stands at 20 ma. In one or two minutes the amount of the current will increase to 25 ma. to 30 ma., and it is kept at this figure for ten minutes. Such an application is made once every two weeks. This method is quite sufficient where the ulceration is confined to the lower portion of the bowel, but where the disease extends higher up slightly more complicated apparatus is necessary. Mr. Wallis and I have had constructed a special electrode for dealing with rectal ulcerations above this point. Shortly, the method is that the lumen of the bowel above the affected area is stopped up by an inflated rubber bag; from this point downwards the intestine is distended with a zinc solution; the electrode is a hollow zinc tube, to allow of the solution being introduced and the rubber bag being inflated; the portion of the electrode in contact with the sphincter is covered by an insulating surface of vulcanite, and if ulceration occurs at this point it must be dealt with before dealing with the upper area.

In the cases quoted above (those of Drs. Webb and Baker) kataphoresis has been employed in dealing with chronic ulceration of the large intestine, which was more or less superficial. Where the ulceration is submucous it is, however, necessary to liberate a sufficient quantity of the metal to allow of penetration into the deeper tissues. Nitrate of silver is not a suitable salt for this purpose, because it is not easily broken up by galvanic currents; zinc sulphate, on the other hand, is easily broken up. Further, the application of the silver salt is followed by considerable pain a few hours after application, whereas the zinc salt is nearly painless.

CASES.

Case I.—L. S., aged 30, was operated upon on January 29, 1907, for an ischio-rectal abscess and fistula. She discharged herself from hospital having a sinus which was healing. Patient appeared on June 25 with obvious ulcerative proctitis commencing just within the internal sphincter; she complained of pain and discharge; kataphoresis was commenced at once and eleven applications were made of ten minutes each every two weeks. The pain and discharge were relieved from the first application and the general health was much improved, and she was discharged with the ulceration cured. The salt used was sulphate of zinc 4 gr. to the ounce, except during the last two applications, when a 2 per cent. solution of silver nitrate was used. After these there was considerable pain and discomfort commencing twenty-four hours later. This was not the case when the zinc salt was used.

Case II.—C. D., a woman aged 44, was a typical case of ulceration of the rectum, with marked infiltration of the submucous tissue for about 3½ in. Her symptoms were the same as those described in the first case, and in addition there was a well-marked stricture about 2 in. from the anus. All this followed on a protracted labour which occurred nine years previously. At St. Mark's Hospital I removed most of the infiltrated tissue, but the free edge of the mucous membrane which was brought to the skin edge was not healthy, and having regard to the former cases I was doubtful as to the result being a complete cure, and the further progress of the case proved the correctness of my surmise. Although the wound healed quite well it was obvious to one who had already had experience of such cases that the infiltration of the submucous tissue was still in progress. As soon as the patient was able to leave the hospital she was sent to Charing Cross for kataphoresis treatment. This woman has had six applications in all, and when last seen her condition was most satisfactory, and I feel satisfied that the further progress of this disease has been stayed.

Case III.—D. E., aged 25, a soldier, was admitted to Charing Cross Hospital, 1906, suffering from ulceration of the rectum and commencing stricture. He had had dysentery in India in 1901, in 1902, and two attacks in 1903. After the last attack in 1903 he had a discharge of pus and blood when the bowels were opened, and he suffered severe pain. The bowels acted from four to five times daily, and he lost over 2 st. in weight. He was admitted to the Netley Hospital in October, 1905, and discharged in March, 1906, and was given antisyphylitic remedies. He went to Richmond Hospital, Dublin, in May, 1906, and received there the same treatment. He had been examined ten times under anæsthesia and the ulcers had been curetted and the rectum dilated. On admission to Charing Cross Hospital a sigmoidoscopic examination was made, when the ulceration was seen to commence about 3 in. up on the posterior wall. The mucous membrane was raised and thickened some distance beyond the ulcer, showing submucous infiltration. The ulcer was scraped for purposes of bacteriological examination. Cultivations produced *Bacillus coli*, scanty growths of streptococci and bacilli of the proteus group. On July 30, 1906, I attempted to remove the ulcerated bowel by a trans-sacral operation. After exposing the bowel at the back it was not thought desirable to proceed with the operation, and the sacral wound was closed and a left inguinal colotomy performed. The patient's general condition improved, but although daily irrigation of the ulcerated bowel was performed through the colotomy wound and the ulcerated surface dressed with mercuric or iodoform gauze the discharge from the rectum continued. Kataphoresis by zinc electrolysis was started on January 31, 1907, and was carried out twice weekly. Marked improvement was seen to occur in the character and quantity of discharge after the second application, and on February 26 the ulceration had healed. There was some contraction of the bowel about 3 in. up, but this was easily overcome by dilatation. On February 27 the colotomy wound was closed. The result of this was a return of the purulent discharge from the rectum. Kataphoresis was recommenced, and the patient was discharged on March 3, 1907. His stools were normal and passed without pain or hæmorrhage, but there was still some slight muco-purulent discharge. The patient went away to Ireland and was seen about four months later. His general health had improved very much, and he gained 28 lb. in weight. There was still some discharge *per rectum*, but this, I felt sure, was due to the persistent daily plugging of his rectum with mercuric gauze, which he had religiously carried out for over four months. When seen in January this year and examined with the sigmoidoscope the ulceration was found to be healed.

Case IV.—S. D., aged 25, a young married woman of good healthy appearance, well nourished, was first seen at St. Mark's Hospital in June, 1903, for a discharge of blood from the rectum which she had had for four years. There was pain on defæcation, and she complained of the bowel prolapsing. She had infective ulcerative proctitis, and was treated by rest and aseptic douches, but refused any operation and left the hospital. She returned in October, when she was treated with injections of silver nitrate and douches of chinosol, and was discharged relieved in November, 1903. She was readmitted on February 8, 1904, with the condition of ulceration much worse and marked submucous infiltration. On February 11 the affected mucous membrane about 2½ in. from the anus was excised and the upper edge was stitched to the skin, but the stitched edge was not healthy mucous membrane, there being a considerable amount of submucous thickening. The wound was obviously septic from the first, as there was an immediate rise of temperature, which lasted for some time, but she was discharged from the hospital two months after the operation almost well. When seen in May her condition seemed satisfactory, but in July she returned with a recurrence of the ulcerative condition posteriorly. The affected mucous membrane was excised, and she made a good recovery and was discharged a month later apparently cured. She was readmitted in March, 1905, with a return of the ulceration, and in May the ulcerated surface was cauterized. She was discharged at the end of May somewhat relieved, but not well, and was advised to go to Charing Cross Hospital for electrical treatment. Nothing more was seen of the patient for over a year, when she was seen at Charing Cross. Her condition locally was rather worse than it had ever been, but her general health was excellent, as indeed it had been all along. She complained of considerable pain when the bowels acted and at other times. Also there was a constant discharge of blood-stained pus from the rectum. Kataphoresis was commenced on March 7, 1907, and ended November 2 same year, and she had the treatment sixteen times in all. From the first application she derived manifest relief of symptoms. The pain disappeared and the discharge almost ceased. When seen by me in November, 1907, there was no evidence of any ulceration, but merely the scar tissue showing the site of the former ulceration.

Case V.—L. G., a married woman, came under my care about the same time as the last patient. Her condition of ulceration was advanced, and extended 3 in. up the bowel; beyond that there was an area of submucous infiltration. After some weeks of expectant treatment, such as washing the ulcerated area with injections of chinosol, peroxide of hydrogen, Condy's fluid, &c., without any marked relief, either in cessation of the discharge or in lessening of the pain, the woman was advised to have an operation with the idea of excising the affected portion of the mucous membrane. A preliminary colotomy was thought advisable, but this the patient would not consent to, and it was only after some weeks consideration that she consented to any operation at all. The ulcerated tissues were excised, and what looked like healthy mucous membrane was brought down and stitched to the anus. This patient did not do well, and although, as in the last case, her general health and physique were excellent, the ulceration steadily continued its course in spite of everything. Later on a colotomy was consented to, and after this had been done the patient was much more comfortable, being free from pain and able to wash the lower bowel through from the colotomy wound. Kataphoresis was commenced in March, 1907, and has been continued up to present date. The improvement was noticed from the first, the pain was relieved, and the discharge was greatly diminished. Owing to the contraction of the fibrous tissue, the introduction of the electrode was a difficult process, and caused some pain. This woman's present condition is that she is free of pain, and there is no further advance of ulceration, but owing to the length of time during which the ulceration had been existing, the destruction of the mucous membrane of the bowel was so complete and the amount of fibrous tissue so great, that any question of recovery with a view of closing the colotomy wound was not to be considered. This is the worst case of the small series of which notes have been given, but even here the result is far more beneficial than any other form of treatment could produce. The usual course of these cases is a continuously progressive one until the ulceration has invaded the sigmoid, when local peritonitis, followed by adhesions of the coils of intestines in the pelvis, ensues, and ultimately obstruction may occur, which necessitates opening the abdomen with usually disastrous results. This woman is enabled to pursue her daily life without pain or discomfort, except such as may arise in connection with the colotomy.

Remarks.

The cases upon which we have tried this treatment of kataphoresis have all been typical cases of chronic infective ulcerative proctitis, the extent of the disease being dependent upon the length of time it had existed. The various kinds of treatment other than the one under discussion are shown in Cases III., IV. and V. When seen early enough the ulceration may be cured straight away by a few applications of this treatment, as in Case I., but when the disease is of longer standing and the ulceration extends further up the bowel (Case V.), or when it is an after-result of dysentery (Case III.), the treatment is much longer and the introduction of the electrode is painful, and it is not always possible to treat the whole of the affected area by the means at present adopted unless resource is had to a temporary colotomy.

It will be noticed that the salt mainly used in the cases treated by us was sulphate of zinc of the strength of 4 gr. to the ounce, and that the method of introducing it was by means of saturated lint, which was wrapped round the positive electrode. The introduction of such an appliance into an ulcerated bowel must necessarily be painful, and we hope that the new form of electrode may be more efficient and less painful.

In Cases I., IV. and V. a 2 per cent. solution of silver nitrate was used for one or two applications, but in each instance when this salt was used there was considerable pain and discomfort commencing some hours afterwards. Such was not the case when the zinc sulphate was used; in fact, one of the points in regard to it was the complete cessation of pain after the first or second sitting. It must be admitted that the silver salt used was considerably stronger than that used by either Dr. Curtis Webb or Dr. Ernest Baker, and there is no statement of any pain following treatment in their cases, such as followed the use of the 2 per cent. silver solution in our cases. On the other hand, the pathological condition of the mucous membrane was very different in our cases as compared with the other two. In the one set the ulceration was definite and there was submucous infiltration, and the tissues around were inflamed and sensitive; on the other hand, in both Webb's and Baker's cases, the ulceration was superficial and slight in character. At the same time it is a question whether the method adopted by these gentlemen is not the best for the application of the solution to the affected tissues, especially when the affected mucous membrane extends some way up the bowel.

All we can propose to offer in this paper is a suggestion, based upon practical experience of various cases, that this kataphoresis by zinc or silver ions is a scientific method for the treatment of these intractable cases of ulcerative proctitis which is well worth a further trial, and our motive in bringing the method forward somewhat early is to enable others to carry out the treatment, which is quite simply done in suitable cases, and later we hope to bring forward further cases, with even better results.

DISCUSSION.

The PRESIDENT expressed the Section's thanks to the authors for their valuable contribution, which, like the last paper, also dealt with a very intractable disease, and in regard to which any observations from Fellows would be very welcome.

Dr. CURTIS WEBB said he had had six cases of ulcerative colitis at various stages, and in no case had he found pain follow the silver treatment. The 2 per cent. mentioned by Mr. Wallis was much stronger than he (Dr. Webb) had employed. His practice had been to use large quantities, 1 to $1\frac{1}{2}$ pints of $\frac{1}{10}$ per cent., injected with the patient lying on the back, slightly inclined to the left side. After preliminary lavage of the bowel, the fluid passed some way up the colon. He had been anxious lest there might be pain and discomfort if he had used greater strengths. In a recent paper published in Paris it was stated that the ions did not penetrate to any appreciable depth by that treatment; and the authors stated that the ions penetrated no deeper than the subcutaneous tissues. Another point which that line of treatment brought into consideration was the treatment of chronic sinuses and general chronic ulcerations by kataphoresis.

Dr. H. K. RAMSDEN asked whether it would not be well to use cocaine before the application of the zinc, for then the treatment would be just as effective and at the same time painless. He had used the treatment for chronic endo-cervicitis with very good results; also in atrophic rhinitis in one case, in which it was very successful. A case of rodent ulcer was much improved under it, but not cured. In the case of the nose he used an ordinary bichromate battery, and covered the probe with cotton wool dipped in a 2 per cent. solution of zinc sulphate, the negative being held, uncovered, in the patient's hand. The same was done in regard to the uterus. The case of atrophic rhinitis was not cured, but that treatment did it more good than anything else. He had not tried it for proctitis, but he was sure there was a good future for the treatment. He had always used cocaine first.

Mr. LOCKHART MUMMERY said the paper was a very interesting one, because the cases with which it dealt were very difficult ones. Only during the last few years had any real attention been paid to bad cases of ulcerative proctitis and colitis, because only since the introduction of the sigmoidoscope could one see the condition which was present and treat it on scientific lines. Mr. Wallis said a good deal of pain was experienced when using silver nitrate, and that he was using a strong solution. But surely when introducing a drug by kataphoresis, the actual strength of the solution employed did not very much matter, as the same quantity of the drug would be driven into the tissues whatever the strength. He asked whether it would not be worth while to try albuminoid salts, such as protargol or argyrol. Also, Mr. Wallis said that in two of his cases colotomy had been done, and Mr. Mummery would like to know how soon after the colotomy the kataphoresis was commenced, as considerable improvement sometimes followed colotomy. Also, was an anæsthetic used, or was the pain not considered sufficient to warrant that?

Mr. SWINFORD EDWARDS congratulated the authors on the paper, which had been very interesting to him as a new departure in rectal therapeutics. He understood that Mr. Wallis had been limiting his trials of the method to cases of infective ulceration of the lower bowel. He did not know whether the cases included tubercular and dysenteric ulceration, though he alluded to follicular ulceration. Did he treat the last-named condition by the method? He understood Mr. Wallis to say, at the commencement of the paper, that cases of infective ulceration of the lower bowel could only be treated successfully, and that not always, by excision. He (Mr. Edwards) was rather surprised to hear that, because excision would not commend itself to him for such a condition; he would be doubtful of getting union by first intention—a very necessary condition. He had seen a considerable number of cases of infective ulceration of the part following operations get well eventually by the usual methods, viz., either by curetting or by the application of escharotics. He asked whether the method described was applicable to ulcerations higher up, in the sigmoid colon. Up to the present he had relied on the operation of appendicostomy for ulcerative colitis of an intractable nature, and he had been very pleased with the results, which results had been verified by sigmoidoscopic examination.

Mr. WALLIS, in reply, said the cases which had been treated by this method so far had been instances of ulcerative proctitis, but one or two had been done since and not included in the paper, but referred to by Dr. Curtis Webb, of bad tuberculous fistulæ with a long sinus. There was also a case of abscess, containing the *Bacterium coli*, which appeared to have no connection with the bowel. There one application of the treatment produced excellent results. Since the paper was written, a young woman came into the hospital with definite commencing proctitis, not due to operation, but to some infection, possibly vaginal. She was absolutely cured by one application of the treatment, and had been in good health ever since, showing that in recent cases the treatment promised better results than in long-standing ones. He was glad to hear that Mr. Edwards cured his cases eventually; in that he was luckier than most surgeons. He (Mr. Wallis) had found extraordinary trouble in getting them to heal. Three or four years ago he read, at the Leicestershire meeting of the British Medical Association, a paper in which were details of four cases, where he excised the infected portion of bowel and brought down healthy bowel, and in each case there was complete cure, and those were the only ones he then knew in which no stricture remained. The treatment detailed was certainly applicable to fistulæ and sinuses in other regions than the buttocks. As to the amount driven into the tissues with solutions of different strengths, he would ask Dr. Bruce to reply to that. An anæsthetic he considered most desirable.

Dr. IRONSIDE BRUCE, in reply to Mr. Mummery, said the amount of the salt driven into the tissues by the current did not depend upon the strength of solution used. One objection to the silver salt was that it was difficult to break up, whereas the salt of zinc was easily broken up by the galvanic current. That accounted for the choice of the latter metal. The pain produced by the silver salt came on two or three hours after the application, not at the time. He had employed cocaine in order to get the electrode introduced comfortably into the rectum, but in connection with the pain caused by the silver salt, the effect of the cocaine had then passed off.

Surgical Section.

June 16, 1908.

Mr. J. WARRINGTON HAWARD, President of the Section, in the Chair.

Reduction of an old Subcoracoid Dislocation of the Humerus by Excavating the Glenoid Cavity through a Posterior Intermuscular Incision.

By W. G. SPENCER, M.S.

THE forcible reduction of old dislocations at the shoulder has been often described as a successful measure, but the use to which the patient could afterwards put the limb, or whether, indeed, he had thereby been predisposed to a recurring dislocation, has generally been passed over without mention. But the employment of forcible measures has frequently caused additional injury, many dying from rupture of the axillary artery. This method would only now be practised in a limited fashion. Some cases, for one reason or another, remain unreduced, the patients making what use they can of the mobility of the scapula.

Kocher's method of reduction has been recently reviewed and defended by Bach.[1]

Excision of the head of the humerus in order to regain mobility forms the subject of a paper by Shield.[2] In it, and the discussion which followed, are mentioned a number of cases, and since then this operation has often been practised. Some form of anterior incision has been adopted; the operation is difficult, for one has to cut freely through displaced and altered structures, which cannot be well identified; the circumflex nerve is always in danger of being injured, also the other axillary structures. There is left a wound cavity in which blood tends to collect and break down. Although the result obtained may be good as regards mobility, there may be little strength in the arm.

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[1] *Deutsch. Zeitschr. f. Chir.*, 1906, lxxxiii., p. 27.

[2] *Med.-Chir. Trans.*, 1888, lxxi., p. 173.

Lord Lister, in his Presidential Address to the Hunterian Society,[1] described two cases of bilateral dislocation for which he had performed arthrotomy. After freeing the head from its muscular attachments, also partly excising one head, he reduced the dislocation by the aid of pulleys. Keetley[2] advocated arthrotomy, and included a full history of the subject. Lister's method of arthrotomy, aided by extension by pulleys, leaves the shoulder with very limited mobility. After prolonged passive movement and massage the patient will generally not be able to raise his arm above the level of the shoulder.

Subcutaneous division of bands and osteotomy of the neck of the humerus have not obtained any vogue.

The method of obtaining reduction through a posterior intermuscular incision by excavating the glenoid cavity suggested itself to me because it seemed an adaptation to the shoulder of the procedure previously employed at the hip. For a traumatic dorsal dislocation of the hip, of five months standing, in a boy aged 7, I made an anterior incision between muscles, and, without injuring any important structure, excised the obstructing capsule and the dense fibrous tissue filling the acetabulum. After making a well-marked cup, the unaltered head of the bone was returned into place by manipulation without using any force. I afterwards showed the boy at a meeting of the Clinical Society with a good return of movement.[3] I cannot find that I have been anticipated in the description of the operation I have adopted on the shoulder, nor in putting forward the considerations which seem to me to support the method.

As to a posterior incision to expose the shoulder-joint, Kocher, in his "Operative Surgery," describes cutting through the acromion and infraspinatus. I have followed his recommendation in the case of tuberculous disease in a child, in which the glenoid cavity was the chief part affected and a caseous abscess was pointing behind. But that is a quite different operation.

Indeed, all writers contemplate old dislocations of the shoulder from the front. The illustration of a dissection in a tract in the Library of the Royal Society of Medicine, by Bonn, "Commentatio de humero luxato," Lugduni Batavorum, 1782, Tabula IV., which I have had photographed (fig. 1), was drawn from a dissected post-mortem specimen.

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[1] *Brit. Med. Journ.*, 1890, i., p. 1.

[2] *Lancet*, 1904, i., p. 207.

[3] *Clin. Soc. Trans.*, 1895, xxviii., p. 293.

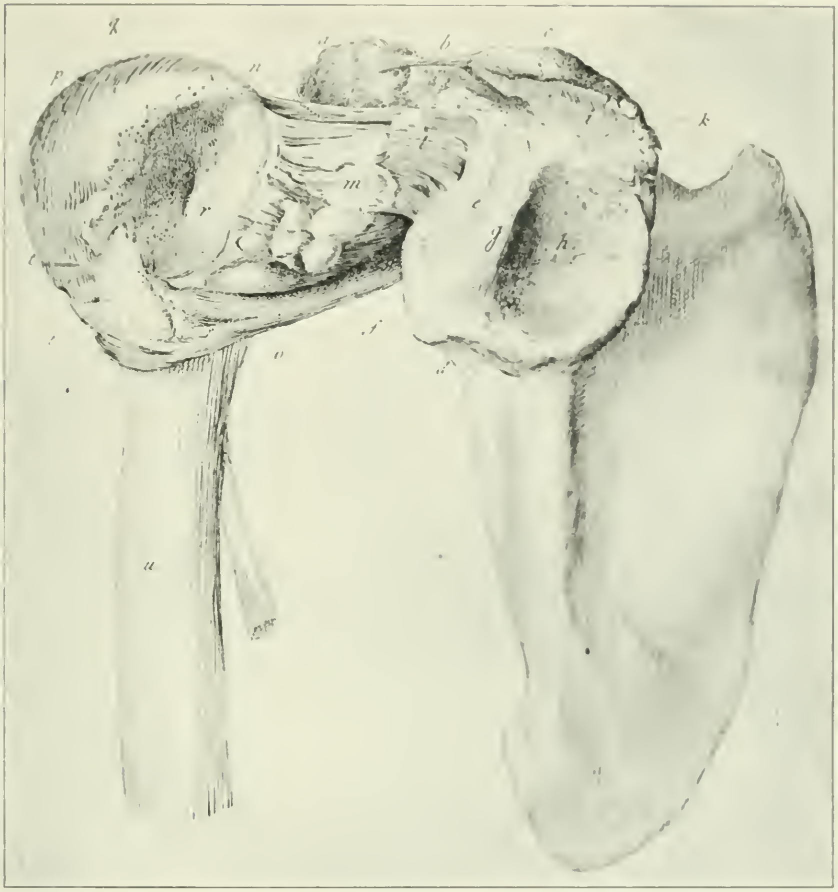

FIG. 1.

Photographed from Bonn, "Commentatio de humero luxato," 1782, Tabula IV.

a	Acromion.
b	Coraco-acromial ligament.
c	Coracoid process.
d	Neck of scapula and site of origin of the long head of the triceps.
e	The original glenoid cavity.
f	The anterior rim of the original glenoid cavity.
g	The posterior rim of the false joint.
h	The false joint cavity under the coracoid process.
i	The superior rim of the false joint under the coracoid process.
k	Anterior rim of the false joint.
l o	The altered posterior part of the joint capsule.
m	Fatty, fibrous, and bony plates originated in blood-clot.
n v	Long head of the biceps tendon.
p	Head of humerus, retaining its cartilage unaltered.
q r s	Eburnated, polished, flattened portion of head, with cartilage absent from contact with the false joint.
t l	Altered anterior portion of the capsule.
u	Shaft of humerus.

It shows in particular the mass of thickened capsule with calcareous plates resulting from the injury, also the remains of the glenoid cavity and the false joint. Moreover, the old shoulder dislocations placed in museums have usually been dissected from the front. I show an exception to this from the Royal College of Surgeons' Museum (fig. 2). It is a post-mortem specimen dissected by Flower, and exhibits the relations of the muscles, but much of the thickened capsule has been cut away, as well as part of the deltoid, infraspinatus and teres minor. The relation of the long head of the biceps and that of the triceps to the dislocation is the chief point to which I would draw attention in the specimen.

Mr. G. R. Ward, who acted as my dresser, kindly made for me two drawings, one (fig. 3) to show the normal position of the muscles behind the shoulder when the arm is outstretched; the second drawing (fig. 4) shows the glenoid cavity as exposed at the operation. The occasion for the employment of the operation was as follows: A weakly man, aged 53, formerly a compositor, had had ten years before a cerebral vascular lesion causing right hemiplegia and aphasia. From this he had so far recovered, but his right side remained weak, and his speech was difficult to understand, which was partly due to his being edentulous. In August, 1907, he was walking on the pavement when he slipped off the kerb and fell into the gutter, hurting his left shoulder. He went at once to a doctor, who, he said, told him there was nothing wrong with the shoulder. Three weeks later, on the advice of a friend, he attended the out-patient department of a general hospital, where he was anæsthetized and an attempt at reduction made. He was then admitted to the hospital ward, and two further attempts at reduction tried, both under an anæsthetic. He was afterwards given to understand that nothing more could be done for him and he left the hospital.

Two months later he went to Dr. Tippett, of Staines, who sent him into the Westminster Hospital under me. His left arm could hardly be moved at all from his side on account of a subcoracoid dislocation, by which the head of the humerus was firmly fixed to the scapula. There was extensive eczema in the axilla and around owing to the arm being kept to the side. This dislocation on the left side, along with the hemiparesis on the right, rendered him quite helpless. Thus an operation to free the left shoulder was particularly indicated, whatever objection might be made to it on account of the weakness of the patient and the probable state of his cerebral vessels.

I operated on November 13, 1907, three and a half months from the accident, after especial care had been devoted to preparing the armpit,

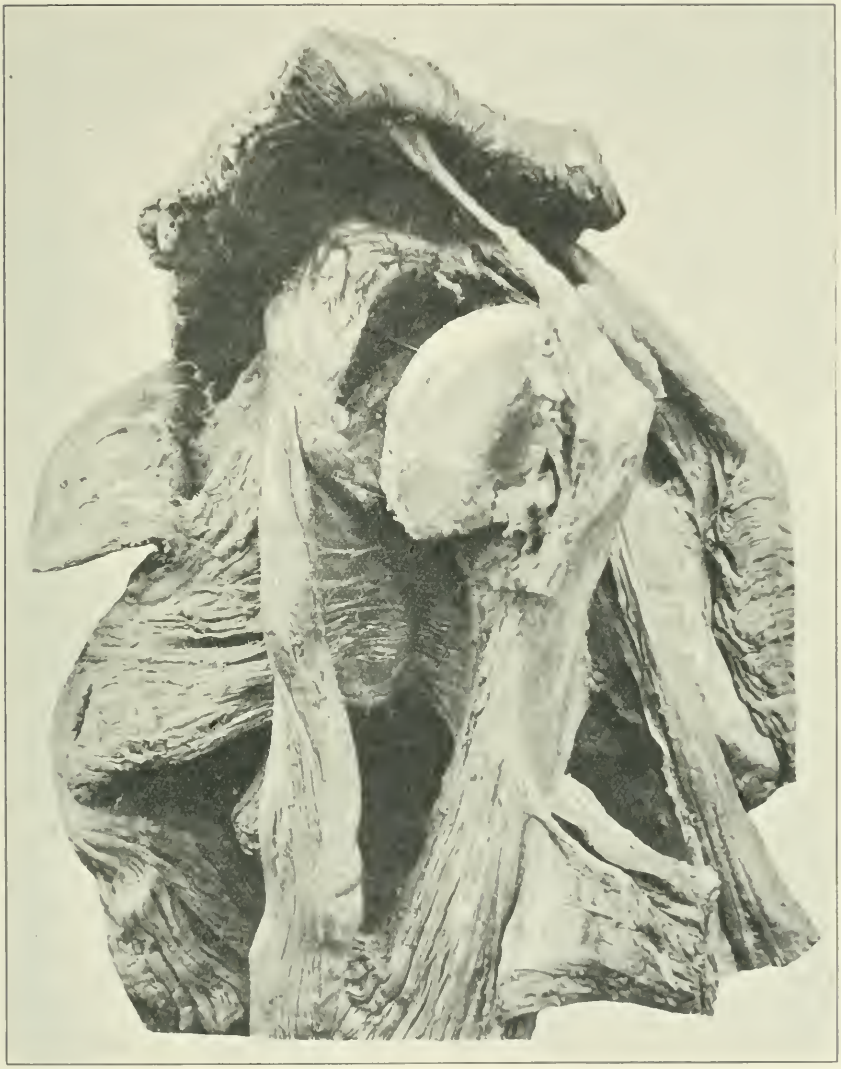

FIG. 2.

Photographed from Specimen No. 1,748 in the Museum of the Royal College of Surgeons of England. A dissection of a post-mortem specimen of old-standing subcoracoid dislocation of the shoulder by the late Sir W. H. Flower.

Part of the deltoid, infraspinatus, and much of the altered joint capsule have been cut away, but the specimen shows the remains of the glenoid cavity, with the origin of the biceps and triceps; also the false joint.

owing to the eczema which had followed upon the adduction of the arm. The patient was laid on his right side, I stood behind him on the left, the dresser held the left arm as much raised as he could, so that the posterior surface of the arm looked upwards and forwards; at the same time he pushed the humerus towards me. This is a primary feature of the method; the glenoid cavity and neck of the scapula can be thus

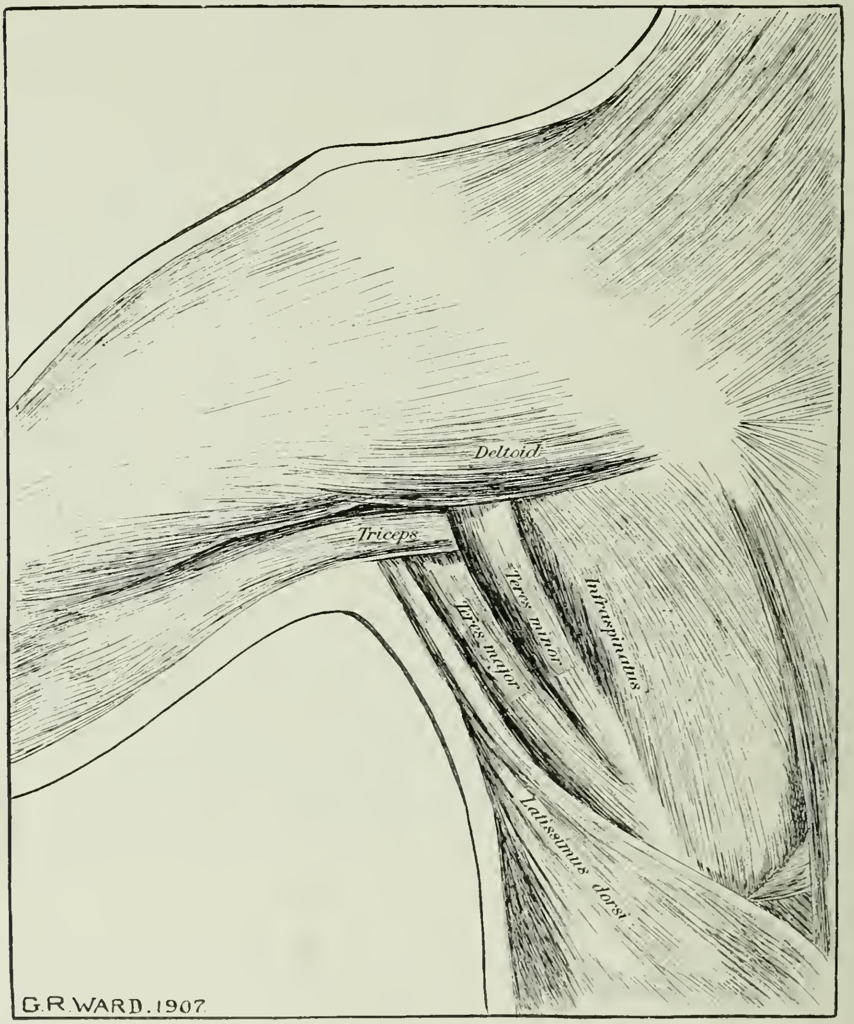

Fig. 3.

The muscles behind the shoulder.

pushed towards the incision and held there, fixed by making use of the head of the humerus in its displaced position underneath the coracoid process. Mr. Ward's second drawing (fig. 4) illustrates this point. A semilunar skin flap was turned upwards over the acromion by making a curved incision, which commenced over the middle of the spine of the

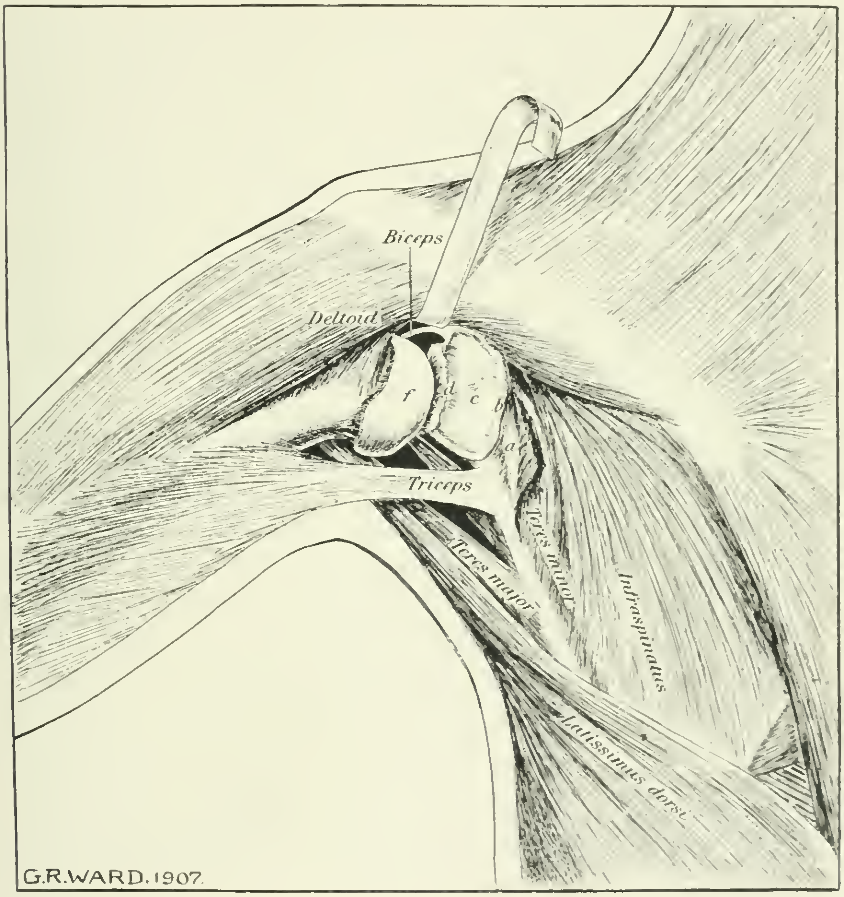

FIG. 4.

Diagram of the exposure of a subcoracoid dislocation of the shoulder from behind.

a Triangular area exposed of the dorsum and neck of scapula.
b Posterior rim of the glenoid cavity.
c Middle of the glenoid cavity.
d Anterior rim of the glenoid cavity.
e Posterior rim of the false joint.
f Head of humerus.

scapula and ended over the posterior fold of the axilla. A photo (fig. 5) taken soon after union shows this line. Then the upper border of the latissimus dorsi was freed and retracted downwards, the posterior border of the deltoid retracted upwards. The next step (figs. 4 and 1) was to raise by means of a raspatory the teres minor and infraspinatus from their origin near their tendons so as to expose the axillary border and a small triangular area of the dorsum and neck of the scapula. This led to a good view of the origin of the long head of the triceps from the lower edge of the glenoid cavity and the axillary border of the scapula. Now I began to cut away piecemeal a tough mass of tissue representing the posterior part of the capsule and to chip away the posterior rim of the glenoid cavity until I had reached the origin of the long head of the biceps from the upper margin. I had then the long head of the triceps below and the long head of the biceps above both well defined at their origin, but uninjured. Between these two tendons was a mass of fibrous tissue and fat, representing the capsule remains of blood-clot, some of which had become altered into melon-seed bodies in the former joint cavity (fig. 1, *l*, *m*, *o*). No actual joint cavity remained, but a section under the microscope of a piece which had been cut away showed synovial membrane and greatly thickened subsynovial tissue. The rest of the thickened capsule was next excised in small bits, progressively with the removal of bone chips from the middle of the glenoid cavity, until the anterior rim, which had come to form the posterior margin of the false joint below the coracoid process, had been removed. When this was done the head of the humerus was exposed. Thus a cup-shaped cavity had been excavated between the origin of the biceps above and the triceps below, and all the altered capsule had been cut away. A manipulation of the humerus by Kocher's method then caused the head to be reduced into this excavation. This manipulation, without using force, had been tried before, but it was only after the complete removal of the anterior rim of the glenoid cavity, including the posterior border of the false joint, that reduction readily occurred. That a false joint had formed was further shown by the escape from it of synovial fluid, whilst none was found at the site of the true joint. After reduction, the head of the bone could be well inspected; its cartilaginous surface was unaltered, its muscular attachments uninjured. When extension was made on the arm, the head of the bone was only pulled downwards just enough to allow of the fingers being inserted between the head and the acromion; when extension was relaxed the head came into contact with the acromion. This was due to muscles and ligaments having shortened.

The head could not be pushed further back than the excavated glenoid cavity on account of the tenseness of the subscapularis; in the excavated cavity the head rotated easily and the arm could readily be carried into the vertical position; on removing the retractors the muscles returned into place, and as the head of the bone now filled the deepest part, no wound cavity remained, so the skin was sutured without a drain. I had expected to meet with the dorsalis scapulæ artery, but I did not cut far enough down the axillary border to wound it

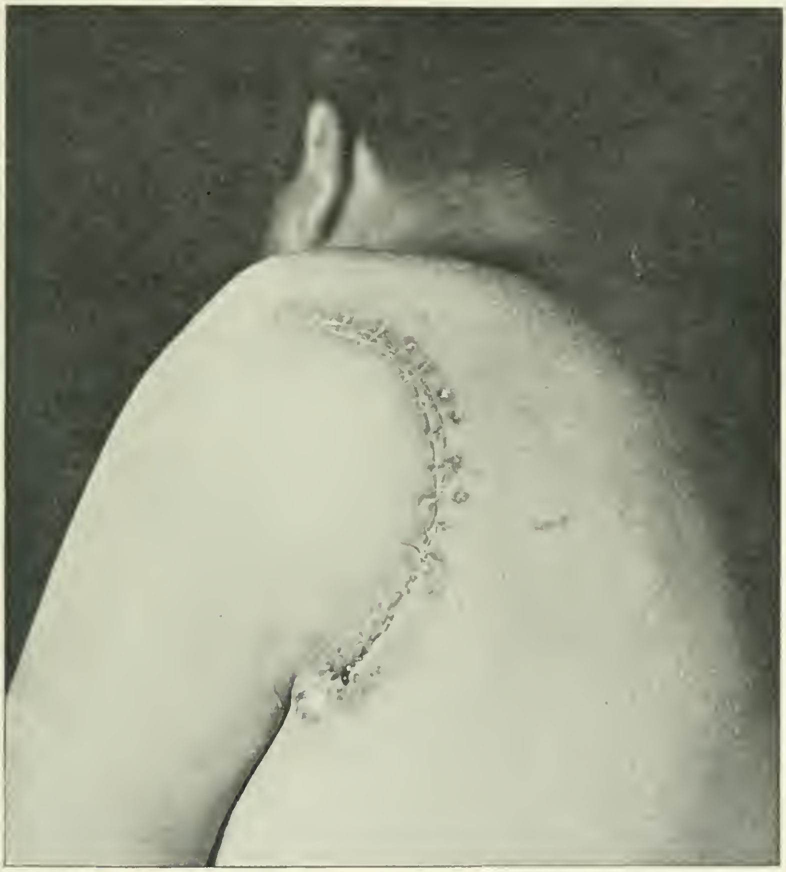

FIG. 5.

Photograph of patient shortly after removal of the sutures, to show the line of the incision.

The whole plan of the operation had kept me far away from the circumflex nerve, and, of course, from the large axillary vessels and nerves. The wound healed well (fig. 5); before the sutures were removed the arm could be readily rotated. As soon as there was firm union, massage, passive movement, and electrical excitation were adopted. The arm could be raised easily to the vertical (fig. 6), and the patient slowly regained the use of the limb in a way which was quite satisfactory considering his general debility.

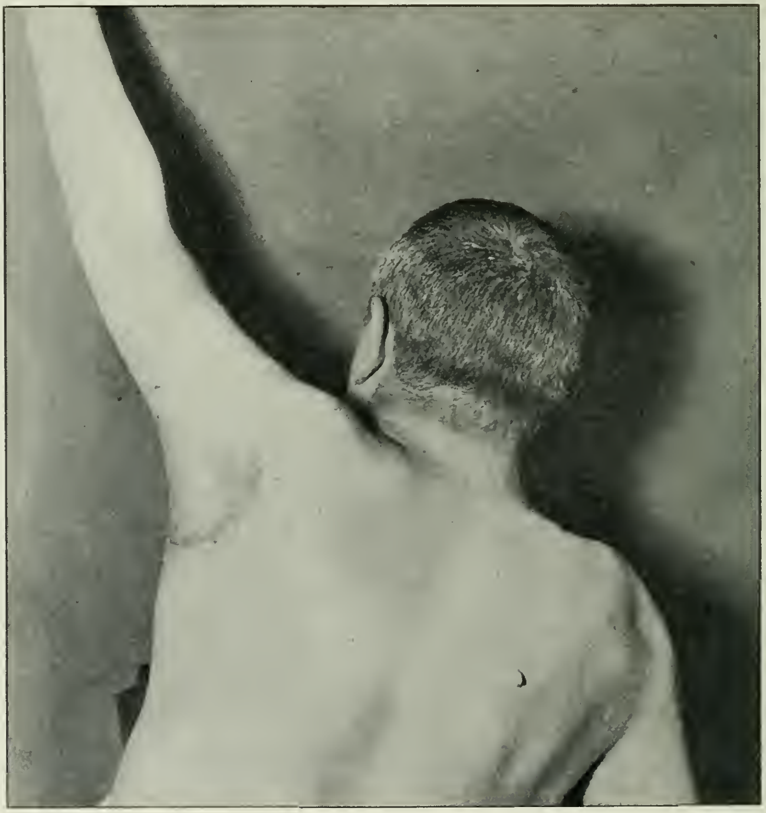

Fig 6.

Photograph of patient two months after the operation, to show the elevation of the arm.

An X-ray photograph taken two months after the operation showed the outline of the unaltered head of the humerus in the excavated glenoid cavity. A relative opacity in the line of the biceps marked a ridge of induration dating from the accident, due to blood extravasation.

CONCLUSION.

This method of treating an old shoulder dislocation appears to have the following advantages:—

(1) The joint is exposed without dividing any important structure, and the muscles which have been drawn aside fall back into place, so that no wound cavity is left in which blood-clot can collect, nor is drainage required.

(2) Through this incision can be removed the impediments to reduction, viz., the altered joint capsule and the bone forming the posterior rim of the false joint, together with the middle portion of the glenoid cavity, without disturbing the important origins of the biceps and triceps from the upper and lower borders of the rim.

(3) At the operation an assistant can cause the deformity resulting from the dislocation to aid the surgeon; by means of the arm he can push the neck of the scapula towards the surgeon, and hold it fixed whilst the glenoid cavity is being excavated.

(4) The head of the humerus is replaced with its muscular insertions and its articular surface intact. It can then move freely in the excavated glenoid cavity; but it is not liable to become displaced again, for all the muscles are still attached, yet have become somewhat shortened in the time which has elapsed after the accident; hence passive and active movements can be begun early. If, indeed, the operation be adopted for a quite recent dislocation, whilst the rotator muscles are still stretched, or perhaps partly torn, then it may be necessary to limit the movements of the shoulder until the muscles shall have contracted up.

DISCUSSION.

The PRESIDENT (Mr. Warrington Haward) said the Section would undoubtedly agree that their best thanks were due to Mr. Spencer, whose paper marked a distinct advance in the treatment of that very troublesome condition, old unreduced dislocation of the shoulder. Most of those present had probably seen very disastrous results from attempts to reduce old dislocations, and the present case seemed to show very well the changes which occurred, even within three and a half months. No doubt most of the dislocations in which disaster had occurred had been of much longer duration. But even in the present case great changes had occurred in the glenoid cavity, and the operation devised by Mr. Spencer, with a posterior incision which avoided the division of the important muscles around the joint, seemed to have given an excellent result.

Mr. RUSHTON PARKER agreed that the result in the case amply justified Mr. Spencer's procedure, but would like to hear, before proceeding, what movement the patient was capable of before the operation.

Mr. SPENCER replied that the arm was absolutely bound down to the side and there was eczema of the axilla all round. He did not attempt any other form of reduction; all that was done before the operation was to try to prepare the axilla.

Mr. RUSHTON PARKER (resuming) said the points which struck him were, first, the amount of utility of limb likely to exist in a case of unreduced dislocation of the humerus; secondly, the amount of benefit that could be derived from attempts at reduction; thirdly, whether benefit could be brought about by operative means. He was sure the present case justified the procedure adopted. He supposed the patient could perform movements strongly and had a capable arm.

Mr. SPENCER reminded Mr. Parker that the patient was a weak old man, was paralysed on one side, and the arm treated was the only one he could use. He had not been able to go to work again.

Mr. RUSHTON PARKER (continuing) said, with regard to previous cutting operations for the purpose, the one which attracted his attention most was that published by Lord Lister—a case where double dislocation of the humerus was operated upon. He, however, was not very favourably impressed with the result, and thought the patient would have been as well, if not indeed better, without the operation. There was a too ready tendency to suppose that unreduced dislocation of the head of the humerus was an unmixed evil. Years ago, when he was much associated with the late Mr. Hugh Owen Thomas, who was such a master on bones and dislocations, he often persuaded patients to put up with dislocations, and he had photographs of them. Some cases of unreduced dislocation of the humerus he had deliberately left alone, and the patients managed fairly well. In one or two cases where he had attempted to reduce not ancient, but comparatively recent dislocations of the humerus, he had failed, and the patient had to put up with the condition—that was after all the means at his disposal had been tried, such as hand traction and counter-pressure on the scapula. The result was not a seriously maimed arm. He had reduced with his hands, and without anæsthetic, dislocation of the head of the humerus three months old, by letting the patient sit up in a chair and getting someone to hold her round the waist, and pulling and manipulating. He asked what Mr. Spencer meant by Kocher's manipulation, as he (Mr. Parker) was taught a mode of manipulation before Kocher's name was heard of, namely, by the late Mr. John Marshall, at University College. The first case of the kind that he saw after returning to Liverpool was a recent case of dislocation, in which he elevated the arm and pulled it back. On another occasion he was in Mr. Thomas's surgery—Mr. Thomas did not seem to believe in manipulation—and he (Mr. Parker) said it was a good case for reduction by manipulation, and he was therefore allowed to try it. It was successful, and there was no mystery about it, but it was not always successful. In these days of aseptic surgery what Mr. Spencer had done was worthy of attention, and the plan deserved following up. Still, he thought the literature of the management of bones and joints was not yet extensive enough to make surgeons acquainted with the degree of defect which could be tolerated without impairing working men from performing their daily duties, or other men getting along in comparative comfort. He did not say that from any reluctance to operate, but thought more attention might be directed to this point. Had it not been foreign to the present paper, he would have discussed the elbow-joint. He knew a joiner who managed to saw very well with his elbow ankylosed at a right angle.

Mr. THOMAS H. KELLOCK said he had been particularly interested in Mr. Spencer's account of his operation, which he (Mr. Kellock) thought he could fairly claim to have anticipated. In the last volume of the *Transactions of the Clinical Society* he recorded an almost identical case. He operated upon a man who was the subject of an old unreduced subcoracoid dislocation of the shoulder, complicated by an united fracture of the neck of the humerus, and it occurred to him that a better result might be obtained by saving the insertion of muscles about the upper end of the humerus and excising the joint by removing the glenoid cavity. That he therefore did. He, however, approached the joint from the front, and he did not think there was much difference in the principle of the operation, whether the approach was from the front or the back. It was comparatively easy from the front, and was done without serious damage to surrounding structures. He removed considerably more of the glenoid cavity than Mr. Spencer had done, and he thought that he (Mr. Spencer) had left more bone than was likely to be of service to the patient; the surfaces must be closely in contact, and he thought there would be a disposition for ankylosis to occur. Mr. Kellock's patient was a blacksmith, and he saw him recently. He had an excellent arm, and although he could not do overhead movements well he could do his garden work and was able to wheel a wheelbarrow, which he (Mr. Kellock) regarded as very satisfactory. The last photograph shown by Mr. Spencer represented almost exactly what happened in his own case. It was a poor elevation, and it was obvious that the scapula was moving with it. He found some difficulty in getting at the front of the neck and the glenoid cavity: and in the paper to which he had referred he had said that if he should get another similar case he would attempt to combine the posterior and anterior incisions. Having heard Mr. Spencer's remarks he thought that would be a more satisfactory way of approaching that deformity; it would give almost perfect command of the joint. He asked what amount of movement in the shoulder-joint of Mr. Spencer's case was perfectly free, because he thought the bones must be almost grating, owing to their proximity to each other.

Mr. Spencer, in reply, said that of course there was often mobility of the scapula in an unreduced dislocation, but those subjects of it whom he had seen had not been able to raise the arm to the level of the shoulder. The arm, however, was a powerful one for all under-hand work. With regard to excision of the head of the humerus, he had done it, and he had seen very good operations of others, which had healed; but he had never seen any such man with any strength in high movements. With regard to Lord Lister's method, Lord Lister himself mentioned that after massage for six months in his cases neither of them could again raise the arm to the level of the shoulder. He had read Mr. Kellock's paper, to which that gentleman referred, and he did not intend that the case now described should be in any sense the same as his. Mr. Kellock used the anterior incision, and therefore must have divided some muscles. He (Mr. Spencer) used the posterior one. Mr. Kellock excised the glenoid cavity through its neck, *i.e.*, he divided the biceps and triceps tendons, two things which, he wished to make it a central point in his paper, he had left. Wheeling a wheelbarrow was under-hand work. The point was whether the man could raise his arm above the shoulder. Perhaps the scapula in his case moved a little, but he thought the elevation of the arm shown in the photograph was a very fair one, and occurred at the excavated shoulder-joint. He was not present when the photograph was taken.

Cystic Tumour of the Suprarenal Body successfully removed by Operation, with Notes on Cases previously published.

By Alban H. G. Doran, F.R.C.S.

Introductory Remarks.

Many observations have recently been published about the pathology, diagnosis and treatment of tumours of the suprarenal body, and of new growths developing in other organs from "rests," as they are termed, of adrenal tissue. An instance of the latter type of tumour was reported by myself last year.[1] In the autumn of 1906 I removed a kidney subject to "hypernephroma," the patient surviving the operation for three months; but there were secondary adrenal deposits, one appearing as a vaginal polypus. Eleven months later I removed a tumour situated in the left lumbar region. It proved to be a cyst of the suprarenal body itself, unilocular and full of a bloody fluid. Henschen would rank it as a *struma suprarenalis cystica hæmorrhagica.* I will now relate my case, and afterwards make some mention of previously reported instances of cystic tumour of the suprarenal body large enough to be of clinical and surgical interest.

The Case.

C. L., aged 62, was admitted into my wards in the Samaritan Free Hospital on October 1, 1907, on account of an abdominal swelling and pain. She had been married for thirty years and had borne nine children, the last confinement occurring eighteen years before admission. There had been no abortions. All the patient's labours were normal except the last, when the forceps was applied. She had never suffered from any puerperal complications, but enteroptosis developed during the later pregnancies. In 1897 she was laid up with influenza, which left her very weak and liable to bronchitis; at the same time she suffered from frequent attacks of pain after food and vomiting. The influenza troubled her again several times; on the last occasion, which was in 1904, she became deaf in the left ear.

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[1] "Malignant Vaginal Polypus secondary to an Adrenal Tumour of the Kidney." *Journ. Obstet. and Gynæc. Brit. Empire*, June, 1907, p. 449; and *Trans. Obstet. Soc. Lond.*, xlix., p. 182.

History of the Present Illness.—The dyspeptic attacks, which had never ceased entirely, became severe last summer, and to them were added sharp abdominal pains, which were at their worst during the night, and were referred to a lump in the left side. She was under the care of Dr. Alexander Davidson, of Cornwall Road.

Condition on Admission.—The patient was fairly well nourished. The abdominal walls were thin, and below the umbilicus extremely lax, forming a flaccid swelling, tympanitic on percussion. There was no evidence of separation of the recti. A firm, oval body, freely movable, occupied the left loin. When the patient lay in bed it retreated for the greater part under the ribs, its lower portion rotating upwards into the epigastrium. It could be pushed downwards and inwards to the extent of over 3 in., until its lower pole lay below the level of the umbilicus; when held downwards there was always more or less resonance on percussing its anterior surface. The right kidney could not be felt. The urine was repeatedly examined; there was no history of hæmaturia, and I never found any trace of blood, but there was always a little rather dense mucous deposit; the secretion was scanty—under 25 oz. in twenty-four hours—and the specific gravity low—as a rule ranging from 1008 to 1022. On October 7 I made a cystoscopic examination of the bladder, with the kind assistance of Mr. Malcolm. The mucous membrane was pale, the right ureteric orifice normal, whilst slightly turbid urine was seen issuing from the orifice of the left ureter. These appearances are worth recording, as they naturally led me to suspect that the left kidney was the seat of the tumour, which was not the case. The tongue was clean, the appetite good and the bowels regular. The pulse was 72, small and regular. The maximum temperature during the first week after admission was 98·8° F. The uterus had undergone senile changes. The menopause was complete by 45. No part of the tumour in the left loin could be pushed down to the level of the pelvic brim. The nature of the tumour was somewhat obscure, and altogether it appeared to be renal.

The Operation.—On October 15, 1907, I removed the tumour, assisted by Dr. William Griffith; Mr. Morley administered the anæsthetic. The patient was placed in the horizontal position. A vertical incision was made through the left rectus muscle, near its outer border, beginning about 2 in. below the costal cartilages and extending 4 in. downwards. I passed my hand into the peritoneal cavity as far as the right loin; the right kidney was in its normal position and was not enlarged. The splenic flexure of the colon lay in front of the lower part of the tumour, which was drawn downwards and exposed by an incision made through the peritoneum on the outer side of the descending colon. The tumour lay in a capsule made up of connective tissue, whence it was easily enucleated in front, outside and behind, without any subsequent oozing. There were several large vessels running into the tumour internally and from above; the tumour now proved to be a thick-walled cyst, the left kidney lay entirely behind and mostly below it; the tail of the pancreas, which I could feel, did not touch the tumour. I secured the vessels with No. 4 China twist close to the cyst wall and fixed on a Doyen's clamp in two places, as the tumour was heavy and threatened to tear itself away from its connections; on dividing the vessels and surrounding connective tissue it was set free, and the tissue included in the two clamps was carefully tied. By the above manœuvres no large vessels were endangered by the application of clamps and ligatures to parts not thoroughly exposed, and no oozing occurred. I pushed up the kidney and sutured the cut edges of the peritoneum with continuous catgut, uniting the muscle and integument with interrupted silkworm-gut sutures. The patient was troubled with cough, and suppuration of the lower end of the wound occurred, but there was no evidence of any effusion or suppuration in the structures whence the tumour had been removed.

On November 21 the left kidney could plainly be defined—it was not enlarged and was quite free from tenderness; it lay almost entirely below the level of the last rib and could be pushed for about 1 in. upwards. The urine was clear, pale yellow, slightly turbid, with a little mucous deposit containing renal cells but no casts; the specific gravity was 1010 and there was no albumin; the daily secretion was rather scanty.

Dr. Davidson has kept the patient under observation since her discharge from hospital. She suffered badly from cough in the winter months, which caused fresh trouble with the cutaneous part of the abdominal cicatrix. By the middle of February the cough had subsided and the patient's general condition was satisfactory.

Note on After-History.—Since I prepared this report the patient came under the care of Dr. Davidson again for attacks of vomiting, which were successfully cured by the end of April by small doses of ipecacuanha, and the constipation and distension associated with the vomiting also subsided. On April 27 I examined the patient; I found that the left kidney was tender to touch, and all of it except the upper pole lay below the level of the last rib. There was no tumour nor hardening in the left loin, abdomen, or pelvic cavity; the enteroptosis had rather increased. Dr. Davidson saw the patient at the end of May in very good health.

Description of the Tumour.—Before the tumour was taken to the College of Surgeons it was accidentally dropped on to the floor of the operating theatre, so that it burst, and its contents, which consisted of about ½ pint of bloody fluid mixed with broken down tissue, were lost. When fresh the surface of the tumour was of a deep purple bronze colour. At the College it was put into a formalin solution, which became deeply blood-stained and was repeatedly changed. Three months after the operation it was placed in a glycerine solution, which soon assumed a pale red tint. Mr. Shattock removed a piece of the cyst wall at the line of rupture and prepared sections for the microscope.

Naked-eye Appearances.—The tumour had shrunk considerably after rupture, but its walls were, from the first, distinctly rigid, so that it simply became smaller without collapsing. When I examined it three months and a fortnight after its removal, its vertical measurement was 4 in., the horizontal 4½ in., and the antero-posterior 3 in. It had lost its purplish tint and assumed the dull reddish brown colour of a cricket-ball. The surface was fairly smooth, except where some tracts of condensed connective tissue were adherent to it; the walls were from ⅙ in. to ¼ in. thick and of tough consistence; the cut surface was dull reddish brown, although uniformly stained with blood, being of precisely the same tint as the outer surface of the tumour. No fibrous or muscular structure could be detected even by the aid of a hand lens, nor were any yellow spots, calcified patches, minute cysts or lacunæ exposed by the section; I could not even detect a blood-vessel. The inner wall was rough from deposits of old clot on its surface; at one or two points the clot was very pale, but there were no yellow patches or tuberosities. The cyst cavity was absolutely single, not a trace of even a rudimentary septum could be found on the inner wall. Thus, to the naked eye, the cut surface of the cyst wall did not show the appearances characteristic of a blood-cyst of the suprarenal body. There was, in fact, no macroscopic indication of adrenal tissue, yet by the aid of the microscope such tissue was readily distinguished.

Microscopic Appearances.—On February 14 I examined, with Mr. Shattock, some sections of the cyst wall which he had prepared for the microscope. The cyst wall proved to be much less homogeneous than was suspected. There was a stroma of fibrous tissue without any plain

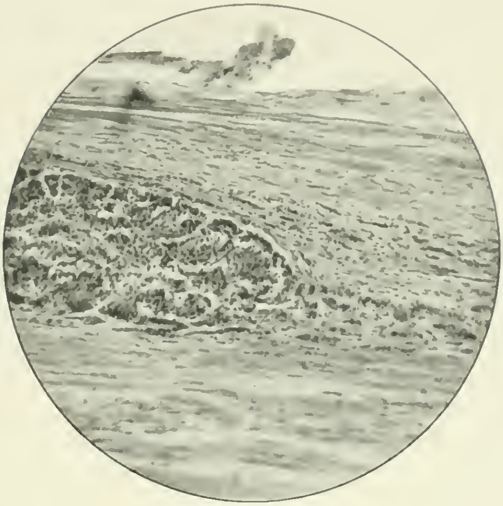

FIG. 1.

Section through the cyst wall showing a nodule of adrenal tissue embedded in the fibrous stroma.

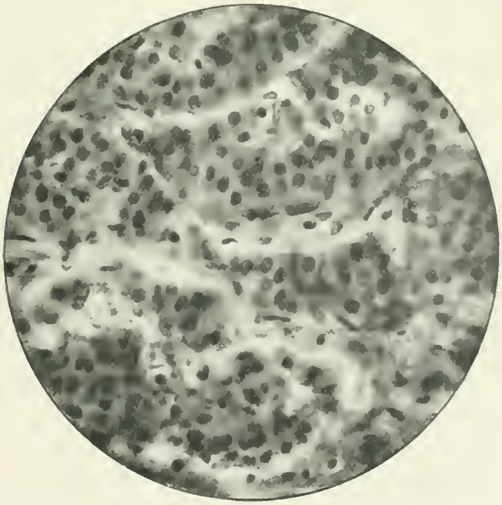

FIG. 2.

A portion of the nodule of adrenal tissue seen under a high power.

muscle; some large veins were detected, but there was no indication of angiomatous nor of lymphomatous tissue; a nodule of adrenal tissue was seen embedded in the stroma towards the outer surface of the wall, whilst deeper in the cyst wall lay a plexus of cells of the adrenal tissue type arranged in a highly atypical manner. There was no epithelial lining to the inner wall of the cyst, but a distinct layer of homogeneous tissue, apparently old coagulum, was observed. Under this tissue was another layer of fibres somewhat denser than in the deeper part of the cell wall. This appearance did not, in Mr. Shattock's opinion, favour the view that the tumour was originally a solid cancer of the suprarenal body and that the interior had broken down. The specimen is now to be seen in the Museum of the College (Path. Series, 3,517 A).

CYSTIC TUMOURS OF THE SUPRARENAL BODY.

I will now make some mention of cases of this kind of tumour already published. By "cystic tumours" I mean cysts of new growths of more than purely pathological interest, mostly blood-cysts, more rarely lymphomas or adenomas, which are true new growths. Several cases which I will relate have already been collected by Henschen and by Terrier and Lecène, but I have made some corrections after reference to the original reports, and added other cases. The first and second are interesting because they were originally published in days long past by able observers who had not the resources of modern science at their disposal. I will therefore relate them at some length. A briefer abstract of each of the remaining cases will be sufficient, as they are recorded by surgeons and pathologists quite recently, in publications to be found in most medical libraries.

Case I.—Greiselius.

The original report of this case, headed "Ren Succenturiatus monstrosus cum ulcere," is to be found in a work published in Leipzig in 1670, entitled "Miscellanea Curiosa Medico-Physica Academiae naturae curiosorum, sive Ephemeridum Medico-Physicarum, Germanicarum," &c., observatio lvi., p. 152 (Rayer misquotes the page). A copy of this work is preserved in the Library of the Royal College of Surgeons of England. Greiselius, of Vienna, seems to have been a good anatomist, and evidently conducted the post-mortem examination of this case with much care.

Contemporary French and German writers presently to be quoted seem justified in admitting this case as a genuine instance of the new growth under consideration. There can be little doubt that the tumour had developed in the suprarenal body, and it was, as in several recent cases, filled with bloody fluid.

Nobilis quidam 45 annorum temperamenti sanguineo-biliosi, post diuturnam Colicam, camq; contumacissimam, generosissimaq; remedia respuentem mortuus, a me apertus fuit, ubi Intestini Coli exteriorem tunicam absumtam, et quasi sphacelatam inveni, ex illâ nempe parte quâ Reni sinistro adjacebat. *Ren* vero *sinister* tantus erat, ut figuram Lienis destruxerit, qui multis tunicis et membranis involutus erat, quibus resectis Ren verus in debita figura et situ inventus fuit: *Ren* vero *succenturiatus tantus* erat, ut totam illam quasi regionem à Diaphragmate (quod una cum Liene ex illa parte clevaverat altius) usq; ad musculum Psoas deorsum occupaverit. In hoc Rene succenturiato erat Ulcus, ita quidem apertum, ut integro pugno transitus pateret. Materia ex hoc ulcere rupto effluxa, erat Aqua rubra ac si bolo armeno tincta fuisset ad libras XII quasi, intus vero adhuc haerens erat densa et glutinosa valdè, fuliginemq; redolebat ad instar carbonum terrae cum summâ nausea et horrore. Haec massa sine materia jam dum effluxa, et particula abrupta ponderabat liber (*sic*) 2 uncias iii. Notandum penes est, quod Nobili huic jam dum filii eodem putatitio Colico morbo mortui fuerint.—*D. Greisel, comm Viennae Dn. D. Jung.*

Thus this patient, "a certain nobleman," aged 45, died after attacks of colic of long duration and of the greatest obstinacy, resisting the most excellent remedies. Colicky pains have been noted in several recent cases; the history of colic in the patient's family is of little value. The report of the autopsy is clear: the left suprarenal body was converted into a big tumour which filled the left side of the abdomen, pushing up the diaphragm and the spleen, and extending downwards to the psoas. In its wall was an *ulcus*, evidently a rupture, big enough to admit the fist. Twelve pounds of red fluid and over 2 lb. of fœtid clot had mostly escaped into the peritoneal cavity. The left kidney was distinct from the tumour.

Traumatism seems highly probable in this case.

Case II.—Rayer

This case has often been quoted, for it was published by a distinguished French physician over seventy years ago, and the tumour is figured in his (Rayer's) fine "Atlas," illustrating his "Traité des Maladies des Reins," plate 54 and plate 55, fig. 3. The surgeon inspecting plate 54 will note how closely the tumour was associated with several inches of the vena cava. The history of injury and pain is very clear.

A woman, aged 75, was admitted into the Charité, Paris, for violent pains in the region of the right kidney. Since 2 years of age she had been lame in the right leg. Within the five years previous to admission she had fallen five times on the right side without any immediate ill effects. Five years before admission she suffered from an attack of agonizing pains in the right loin running down to the pelvis, so that uterine disease was suspected. It is not stated whether this attack occurred before or after the first fall. A few milder seizures of the same kind followed, and another as bad as the first came on three months before admission. There was vomiting, which persisted and became very obstinate, and the pain extended to the right thigh. A tumour was observed about a month before the patient came under Dr. Rayer's care, and the lower extremities became œdematous. The patient's skin was of a greenish yellow tint. There was a large tumour in the right flank, hard in its upper part, where it seemed continuous with the liver, and fluctuating below. There was no tenderness on pressure. The patient died in hospital, long before the days of renal surgery.

The tumour weighed 4 lb.; it pushed up the liver and descended into the right iliac fossa. It contained 1½ lb. (*une livre et demie*, not *litre*, as in some second-hand reports) of black, liquid blood. The kidney, greatly flattened and altered in shape, was found entire and adherent to the posterior aspect of the tumour; the renal tissue and ureter were normal. The left suprarenal body showed no sign of disease; the corresponding kidney was the seat of chronic inflammatory changes.

The above account is from Rayer's "Recherches anatomico-pathologiques sur les capsules surrénales (Capsulæ atrabiliariæ)."[1]

Case III.—Chiari.

A man, "over 60 years of age"[2] and very corpulent, died of heart disease. The place of the right suprarenal capsule was occupied by a spherical cyst nearly 6 in. in diameter. It contained old coagulum, its walls were thin and included circumscribed collections of the cortical tissue of the suprarenal body. No elements indicating a neoplasm could be found in the cyst, which was separated from the kidney by loose connective tissue. The left suprarenal body showed no signs of any change, save senile degeneration. There was no bronzing of the skin, and the existence of the tumour had never been suspected during life.

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[1] *L'Expérience*, November 10, 1837, i., p. 17.

[2] Misprinted "68" in some second-hand reports.

Case IV.—Routier.

Woman, aged 35. Three years epigastric pain and vomiting. Six months[1] tumour observed in left hypochondrium, extending from under ribs down to iliac fossa, dull on percussion, fluctuating at one point. Operation: Median incision, retroperitoneal tumour discovered holding 1,600 grm. of brown fluid. Relations not definable, deep adhesions, drainage, cyst wall tense, its sutures tore away; fatal peritonitis. Cyst found replacing suprarenal body, villous-like growths on inner wall composed of adrenal tissue.

Case V.—Pawlik.

Woman, aged 40. Two years; fall from a ladder, followed by abdominal swelling. Spherical, tense, elastic, fluctuating tumour, descending colon in front. Operation: Incision to left of umbilicus, cyst containing 17 pints of bloody fluid enucleated excepting a small piece left on a kind of pedicle close to vertebræ. Kidney seen on inner side of lower pole of the cyst. Recovery. A small piece of unaltered suprarenal capsule ran on to cyst wall. The tumour was defined as a large hæmorrhagic cyst of the left suprarenal capsule. Its wall included islets of adrenal tissue.

Case VI.—Triepcke and Bier.[2]

Woman, aged 69. Tumour size of adult head, right side of abdomen. Incision along outer edge of rectus; cyst tapped, $3\frac{1}{2}$ pints of turbid fluid with coagula; drainage of cyst cavity, which was first scraped with the curette; death soon after operation from "shock." The cyst, which proved after death to be easily enucleable, occupied the place of right suprarenal body, kidney pushed downwards into iliac fossa. In cyst wall, adrenal elements associated with microcystic degeneration.

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[1] "Six" in the original report, misprinted "dix" in Terrier and Lecène's monograph.

[2] Triepcke: "Ueber Blutcysten in Nebennierenstraumen." I have not been able to procure or see a copy of this thesis; the above is quoted from Henschen, and Terrier and Lecène.

Case VII.—Oberndorfer.

Man, aged 34, no symptoms; death from intestinal obstruction. Round tumour, size of small apple, replaced greater part of left suprarenal body, the unaltered part capping the tumour, a thin-walled cyst which contained clear, pale yellow fluid; lymphangiectasis[1] of remainder of suprarenal body. Right suprarenal body normal.

Case VIII.—Marchetti.

Woman, aged 50. Died in hospital of purulent peritonitis; uncertain origin, but not connected with tumour, which was tense, elastic, fluctuating and situated in region of right kidney. Autopsy: Cyst bilobed, remains of right suprarenal body ran into its wall above; vertical diameter of cyst 4½ in., contents thick, pale yellow fluid; a complete fibrous septum internally. Cyst adhered to vena cava. Adrenal tissue in cyst wall and septum. Compensatory hypertrophy left suprarenal body.

Case IX.—Henschen.

Woman, aged 41. Twenty years pleurisy, from then attacks of pain in left hypochondrium, with vomiting. Three years puerperal thrombosis. During attack of acute rheumatism, big, tense, smooth tumour discovered, extending from left hypochondrium to loin and pushing ribs outwards. Operation (Krönlein): Left pleura tapped, much chocolate-coloured fluid; oblique incision under border of ribs to loin; tapping of cyst, chocolate-coloured fluid as in pleural cavity. Complete enucleation; adhesions to diaphragm and tail of pancreas, inferior mesenteric vein damaged and ligatured. Left kidney lay internal to cyst. Gauze drainage. Death fifth day, from severe thoracic complications. Tumour a unilocular cyst; on inner wall opaque yellow deposits consisting of adrenal tissue.

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[1] Bossard reports a case of lymphangioma cysticum of the right suprarenal body, discovered at an autopsy on a woman, aged 25 (*see* Henschen).

Case X.—Terrier and Lecène.

Woman, aged 52. Four years constipation and attacks of pain in umbilical region, mostly to *right*. Oval, smooth, distinctly fluctuating tumour, size of ostrich's egg, in *left* loin. Operation (Terrier): Median incision above umbilicus. Peritoneum external to descending colon incised; enucleation of tumour easy, only half of it removed, being taken for a pancreatic cyst; base drained. Left kidney found to be distinct from cyst. Contents of cyst lemon-coloured fluid. Walls thin, contained suprarenal tissue. *Right* parotiditis fifth day; recovery; no fistula in cicatrix.

Case XI.—Bosanquet.

Woman, aged 56. In hospital for carcinoma of stomach. Freely movable, firm, rounded tumour below left costal cartilages; not tender on pressure. Fatal hæmorrhage from malignant ulcer (spheroidal-celled carcinoma ventriculi). Tumour an almost spherical cyst, over 3 in. in diameter, in front of left kidney; descending colon on its outer side. "The left suprarenal body was attached to the upper and back part of the tumour and looked normal"; author defines tumour as "cystic adenoma *of* adrenal." I will return to this interesting point further on. Cyst wall thick and fibrous, lined with cells of the adrenal type; contents a semi-fluid, turbid orange jelly, evidently mucoid degeneration of the adrenal tissue. Right suprarenal body bore small white nodule, an adenomatous growth showing fatty or early mucoid degeneration.

Case XII.—McCosh.

Woman, aged 45. Three years dull pain radiating from left loin. Severe attacks of lancinating pain. Slight bronzing of skin. Smooth, globular, elastic, fluctuating tumour in left side of abdomen, pushing out ribs; colon on its inner side. Operation: Oblique incision, complete enucleation of universally adherent cyst, attached to aorta and bodies of lumbar vertebræ; contents 9 pints of dirty yellow fluid. Left kidney much displaced downwards. Forcipressure and drainage; recovery; bronzing of skin disappeared. Wall of cyst thick, containing distinct adrenal tissue.

Case XIII.—Author.

Related above.

Doubtful Case.—Lockwood.

Woman, aged 20. Two years painless swelling, slow growth. Freely movable tumour, size of ostrich's egg, hard, tense, in left hypochondrium, reaching downwards to level of umbilicus; colon defined to its outer side. Operation: Incision outside left rectus, easy enucleation after incision through descending meso-colon; end of duodenum adhered to inner side of tumour. Ureter lay behind tumour. "Recovery" (private correspondence). Thick-walled cyst, fibrous tissue, inflammatory changes, "no glandular structure of any kind could be discovered." Contents, altered blood-clot.

Remarks on Lockwood's Case.—Considered from a clinical and surgical standpoint, this cyst reminds us of several of the blood-cysts in the above series. In outward appearance it closely resembles the tumour which I removed and which is preserved in the Museum of the College of Surgeons, and it also seems very like that described and figured by Henschen. I may refer the pathologist and surgeon to Mr. Lockwood's specimen, which is to be found in the Museum of St. Bartholomew's Hospital, Pathological Series, No. 3,372*a*. The thick wall, blood-stained as in my case, and the single cavity containing "a chocolate-coloured, semi-fluid mass," are features very distinct in the "hæmorrhagic suprarenal cyst." Lockwood himself, referring to the researches of Weldon, Janosik and Rolleston, implies that his cyst might have originated in the suprarenal body, which organ is probably developed from, and is certainly down to a late period of intra-uterine life continuous with, the front part of the Wolffian body. I cannot help suspecting that there may be, in the walls of Lockwood's cyst, some adrenal tissue which has been overlooked. The pathologist doubtful about the homologies of Lockwood's important case should study that surgeon's original report in conjunction with Henschen's well-known monograph.

Mr. Lockwood informs me that about a year ago he removed a similar tumour which adhered to the *lower* end of the right kidney. The same authority describes, in the report above quoted, a case of *multilocular retroperitoneal cyst* removed by Mr. Bowlby, who tells me that the patient was free from recurrence nine years after the operation. It resembles a case of a similar cyst presented by Dr. Bantock to the Museum of the College of Surgeons (Pathological Series, 303*a*). The splenic flexure and descending colon lay on its surface. The patient is living, twenty-two years after the operation for its removal. Mr. Shattock has kindly examined sections from the wall of Bantock's multilocular cyst, but cannot find any adrenal elements. This case and Bowlby's are even more doubtful as to their nature than Lockwood's, as far as origin from the suprarenal body is concerned.[1]

SUMMARY.

Surgical Pathology.—The blood-cyst is not a true new growth: it owes its origin to hæmorrhages into the medullary substance of the suprarenal capsule. There was a history of injury in Rayer's and in Pawlik's cases, which was very probably the cause of hæmorrhage in either one or both; perhaps some pathological change within the organ contributed to the development of the blood-cyst. Such changes, on which it is not necessary to dwell, are probably the sole cause in the majority of cases. The operator, should he recognize the true character of the tumour during the operation, need not search for any extension of disease in its vicinity, and when he can make sure that it is a blood-cyst he need not fear recurrence.

It is certain that adenoma, lymphoma and other new growths seldom convert the suprarenal body into a cystic tumour of interest to the surgeon. Henschen gives in his monograph a good synopsis of the pathology of these cysts, of which there is in the above series one instance of lymphangioma (Oberndorfer) and one of cystic adenoma. Marchetti's bilocular cyst and Terrier and Lecène's big cyst might have owed their origin to old hæmorrhages, as pale yellow fluid is often seen in very old blood-cysts elsewhere.

Those who are interested in the genesis of cysts of the suprarenal body will find much valuable information in the writings of H. D. Rolleston, Ogle, Raymond Crawfurd and Charlewood Turner. The latter writer reports an instance where there were also cysts in the cerebellum, liver and kidney; the adrenal tumour was, I must add, of the size of a fist, but the patient died of the cerebellar disease and the primary seat of cyst formation was not evident. One point very much to our purpose was clear: the cyst in the suprarenal body was certainly not due to hæmorrhage.

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[1] I have recently published a clinical report of these two cases, with notes on Monprofit's account of the removal of a Wolffian cyst: "Cases of Multilocular Retroperitoneal Cysts in Women," *Journ. Obstet. and Gynæc. Brit. Empire*, 1908, xiii., p. 257.

Lastly, I may turn the reader's attention to a preparation which is to be seen in the Museum of the College of Surgeons (Pathological Series, 3517), taken from a woman, aged 55, who died after ovariotomy. It shows "a suprarenal capsule in section with a large, rounded mass in its substance. The remainder of the capsule is distended into a cyst. The enlargement is due to a hypertrophy of the cell columns of the suprarenal capsule, with fatty degeneration of the contained cells." I may add that this report was made by Dr. Goodhart. The preparation may, I think, explain how, in a case of cystic disease of the suprarenal body, the greater part of that organ may be found on the cyst wall, as in the tumour included in the above series, described by Bosanquet.

Symptoms and Diagnosis.—The number of cases of cystic tumour of the suprarenal body remains small, yet the above records show that it gives rise to fairly definite symptoms. I have already noted that a history of *injury* has been obtained in more than one case. *Pain* appears to be the rule; it usually assumes the characters of dyspepsia or fits of colic and leads to the discovery of a tumour. In my own case it was very definite, and the above abstracts show that distinct pain was specified in those reported by Greiselius, Rayer, Routier, Henschen, Terrier and Lecène, and McCosh, making in all seven, to which we may safely add, as an eighth, Bosanquet's case, where, as the original report explains, this subjective symptom seemed mainly, though not entirely, due to coincident malignant disease. The *tumour* in this respect differs from a simple hydronephrosis, although attacks of renal colic may be associated with the latter. Fluctuation seems far less marked than in hydronephrosis, nor does the cyst descend so readily, as the suprarenal body is more firmly supported than the kidney or, we must add, the spleen. In my own case the cyst was, I admit, freely movable, but it always slipped up again when drawn down and did not naturally lie well below the ribs under the abdominal wall, after the fashion of renal and splenic tumours of its own size. The cystoscope may aid in diagnosis, as will be seen in the original report of Pawlik's case. The descending colon is usually anterior to the cyst, but that point is not always accurately indicated. Other symptoms seem far less marked, whilst one, so familiar in association with another disease of the suprarenal body, deserves special notice.

Bronzing of the Skin.—This well-known symptom of Addison's disease was only observed in McCosh's patient. It was slight, yet distinct, and disappeared soon after the operation. McCosh's experience seems to be unique; Henschen, who wrote before the case was published, declares that this "classical symptom" is always wanting (*immer fehlte*) in cases of benign cystic suprarenal tumours, and is absent, "almost without exception," in patients subject to other tumours of the same organ. I may add that in my own case of malignant vaginal polypus secondary to an adrenal tumour of the kidney there was distinct bronzing of the skin during the patient's last days, three months after the operation. The left suprarenal body was found to be free from new growths or any other visible morbid condition. The state of its fellow remains uncertain—it was not found at the autopsy. I mention this case because it shows that cutaneous bronzing, which Dr. W. T. Evans informs me was much more distinct in this instance than my own report could lead the reader to believe, may be present in a subject where one suprarenal body is healthy and where there is no evidence of Addison's disease. None of the cystic tumours described in this communication were bilateral.

Surgical Treatment: Results of Recorded Operations.—Without doubt the right treatment for a cyst of this kind is removal by operation. It should be enucleated from the kind of capsule of connective tissue in which it lies. The suprarenal body is normally kept in its place by fascia which separates it from the capsule of the kidney and holds it well up, far back in the loin. Hence the suprarenal body does not descend with the kidney when the latter becomes movable. When the suprarenal body, on the other hand, becomes converted into a heavy cystic tumour it descends along the outer border or anterior surface of the kidney, stretching its supporting connective tissue, which forms a capsule. The operation essentially consists in the enucleation of the cyst from this capsule.

Diagnosis is difficult; if the cyst be taken for a renal tumour and exposed through a lumbar incision, enucleation might be effected with ease and safety, but in some of the above cases that incision would have proved very unsatisfactory. Therefore the cyst is far more safely dealt with if exposed by a vertical incision through the outer margin of the rectus, as Mayo Robson recommends in operations on solid tumours of the suprarenal body. It allows of efficient exploration, and the above series of operative and post-mortem experiences teaches us that in dealing with a tumour of this kind exploration should be very efficient, seeing that the cyst may adhere to the vena cava, aorta or pancreas.

Incision and Drainage.—Experience teaches us that this incomplete procedure is unsatisfactory. The cut edges of the cyst have been fixed to the edges of the abdominal wound ("marsupialization"); unfortunately the cyst walls, though thick, are not tough like those of the more familiar pelvic and renal cysts. In Routier's case the sutures cut through the tissues, so that the cyst retracted and some of its contents escaped into the peritoneal cavity, with fatal results. In Triepcke and Bier's case it was found after death that enucleation would have been easy. Terrier encountered no difficulty when he enucleated the anterior portion of his cyst, but, suspecting that it was pancreatic, he refrained from completing the process and "marsupialized" the base. The patient recovered, but convalescence was retarded by inflammation of one parotid.

Complete removal by enucleation should always be undertaken if possible. It may be attended with dangerous complications. I have related how Krönlein, in the case reported by Henschen, bravely completed a very difficult operation, but the patient was the subject of pulmonary disease of very long standing, with fatty degeneration of the heart and sclerosis of the coronary arteries, so that the fatal result was not surprising. McCosh's tumour was attached internally to the wall of the aorta. When the connective tissue capsule was incised to allow of enucleation very large vessels were divided. Some lay so deeply that they could not be ligatured; three long artery forceps were applied to them and left on for a time. We are not informed how long after the operation the forceps were removed. The patient recovered. The dangerous proximity of the aorta in McCosh's case reminds us of the observations of Rayer and Marchetti on subjects in the post-mortem room. Both these writers publish drawings of their cysts, which were in the right suprarenal body; they were closely connected with the vena cava.

Pawlik had to deal with a kind of pedicle which ran inwards towards the lumbar vertebræ. It was not secured without much difficulty, and when it was divided, after ligature, a piece of cyst wall as big as a shilling remained on its proximal portion, which receded so far that the operator feared to draw it down in order to excise the fragment. The patient recovered. The after-history of this case and of Terrier and Lecène's, where still more of the cyst wall was left behind, would be of interest.

In my own case enucleation was unattended by any difficulty; I was careful to apply the pressure forceps to all large vessels within sight, avoiding the dangerous practice of pinching tissues in the dark. The surgeon operating on a tumour in the lumbar region is liable to assume that it is renal, and this assumption may induce him to fix a clamp forceps on a part of the aorta, vena cava, pancreas or intestine when he is under the impression that he is simply securing the renal vessels. Lockwood found no difficulty in enucleating his cyst of doubtful origin, although the small intestine adhered to its wall.

In conclusion, I may observe that the pressure forceps, that invaluable invention of Koeberlé, generalized by Spencer-Wells, must be the sheet-anchor of the surgeon engaged in enucleating a cyst of the suprarenal capsule.

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DISCUSSION.

Mr. LOCKWOOD wished to acknowledge the valuable work which Mr. Alban Doran had done in placing these cases on record. Mr. Doran had alluded to one which he (Mr. Lockwood) had described in vol. xlix. of the *Transactions of the Pathological Society of London*, 1898, p. 182. This communication was entitled "A Retroperitoneal Cyst, supposed to have originated in the remains of the Wolffian Body." This was probably the earliest case recorded in this country, and its origin was attributed to remains of the Wolffian body. Since then he had operated upon two other cases of retroperitoneal cyst, both on the left side and both in women. The first, of which an account follows, throws no light on the pathology of these cysts, but the second affords much additional evidence as to their mode of origin. Case 1: This was one of the usual character. The patient was a young woman, aged 29, single, who was admitted into St. Bartholomew's Hospital under Dr. Herringham, suffering from abdominal pain, which was referred to the right side of the abdomen. However, on examination, a rounded swelling about 3 in. in diameter was felt to the left of the umbilicus beneath the left rectus. This tumour was tense and could be pushed back into the lumbar region. It also moved with respiration and was free from tenderness. It was removed through an incision to the left of the umbilicus. As usual, it was situated behind the descending meso-colon. Some large veins crossed over it. Its walls were exceedingly thin and it was attached to the peritoneum by a long, thin stalk, which was transfixed, ligatured and divided. The patient made a rapid recovery, and was quite well a year afterwards. The Pathological Department reported that the cyst wall consisted of fibrous tissue and had no epithelial lining. Some small hæmorrhages had taken place in its loose connected tissue. The fluid contents consisted of slightly blood-stained fluid, so that it was about the colour of straw. The specific gravity was 1010, and it contained about 2 per cent. of albumin. With the exception of some blood-cells, no formed elements could be found in it. This case is so exactly like the one described in the *Transactions of the Pathological Society of London* that it does not call for further comments. Case 2: A young unmarried woman, aged 30, discovered accidentally, three months before her admission into St. Bartholomew's Hospital, that she had a swelling in the left side of the abdomen. The swelling gradually increased. Her chief reason for coming to the hospital was that she was liable to attacks of increased frequency of micturition. On admission a firm, rounded tumour was felt in the left flank. It was freely movable and could be pushed up and down from the situation of the left kidney into the left iliac fossa. It moved with inspiration; the rest of the abdomen was natural. Some urine was obtained by the use of a Luys' separator, and both kidneys were functioning in the same manner, but the urine from the left contained a trace of albumin. This led to the suspicion that the tumour might be renal in its origin, but at the same time the possibility of a retroperitoneal cyst was discussed. The operation was performed through an incision in the left side of the abdomen over the most prominent portion of the swelling, and a cyst with walls about ¼ in. thick was brought in view by dividing the layers of the descending meso-colon. The cyst was spherical and about 3 in. in diameter and tightly distended with a thin, green-coloured fluid: it was attached by a broad base to the lower end of the left kidney. At its attachment to the kidney some amount of solid growth was seen infiltrating the kidney substance. The whole was removed by incising the kidney by two oblique cuts. These did not open the hilum of the kidney, and the incision was afterwards brought together with catgut sutures. A drainage-tube was left in and gave exit to some blood; but, after it was taken out, it was supposed that a little urine ran along the tract. The wound healed by first intention and the patient was quite well six months afterwards. The pathological report is as follows: "The growth is a suprarenal adenoma, in places infiltrating the kidney. The cyst wall consists of fibrous tissue and has no lining of epithelium. A section of the kidney in the neighbourhood of the cyst shows no growth." In this case the cyst had the exact characteristics of the other retroperitoneal cyst, but it had, in addition, an adrenal addition which proves its Wolffian origin. Furthermore, its attachment to the lower pole of the kidney is conclusive evidence that it could not, as has been suggested, have originated in the adrenal body itself and then have gravitated to a position below the kidney, such as that occupied by the other retroperitoneal cysts. Doubtless some of the cysts of the adrenal body collected by Mr. Doran originated not in embryonic remains, but were the result of hæmorrhage, some, indeed, having been caused by violence.

Mr. ALBAN DORAN, in reply, said he was much obliged for Mr. Lockwood's opinion, and was glad he had some more cases to report. He, however, in maintaining that his own tumour arose from the suprarenal body itself, did not rely on the one fact that Mr. Shattock found suprarenal tissue in the walls of the tumour. Several cases described as cysts of the suprarenal body were reported where the patient died unoperated upon, and the relations were clearly made out after death; indeed a portion of the suprarenal body unaltered had been detected on the surface of the cyst in more than one case; and in 1670 it was manifest that Greiselius had no prejudices about this question, because he knew nothing about adrenal tissue. But Greiselius described and chronicled correctly a condition about which he knew nothing, and he had sufficient knowledge to recognize that the tumour was above the kidney and was a cyst full of blood. Rayer's case was carefully reported and figured, and he (Mr. Doran) operated himself, and in detaching the capsule of the tumour, which he now exhibited, a huge vein appeared accompanied by a very respectable artery, which ran towards the great vessels. Therefore he concluded it was a suprarenal capsule converted into a blood-cyst. It should be remembered that in breech presentations (Herbert Spencer) and in cases of whooping-cough in children, blood had been often found freely effused into the suprarenal capsules, and, on the other hand, in two cases of blood-cyst there was a distinct history of an accident. So he (Mr. Doran) thought that there was sufficient evidence that, in all probability, this particular tumour was a blood-cyst of the suprarenal capsule itself. He admitted, however, that adrenal rests might develop above the kidney, and that, as Mr. Lockwood suggested, his or some other of the cases reported might have originated in such a rest rather than in the suprarenal itself. He hoped members present would inspect both specimens—Mr. Lockwood's in the Museum of St. Bartholomew's Hospital and his own in the Museum of the College of Surgeons, for they were remarkably alike. He thought it possible that even Mr. Lockwood's cyst might be a heavy unilocular tumour of the suprarenal capsule which had slipped downwards and inwards behind the peritoneum. He agreed, however, that multilocular retroperitoneal cysts could not be derived from the suprarenal body itself, although they might have developed from rests.

Case of Severe Compound Fracture of the Arm.

By Rushton Parker, F.R.C.S.

A man, aged 44, was brought to the Liverpool Royal Infirmary on February 19, 1907, by Dr. O. Bowen, of Everton, Liverpool, on account of a severe compound fracture of the right arm, which had been jammed between a cart and a wall on the horse taking fright. Dr. Bowen had protected the limb with dressings and a splint. Under an anæsthetic at hospital the lower half of the arm was found to be laid open on the outer side, with a smaller wound on the inner; the humerus was broken across in its lower fourth, splintered, cracked, and stripped bare of muscles. The brachialis anticus and outer head of triceps were torn completely across, the fasciæ widely opened and stripped, exposing the whole interior of the limb in that region. A portion of the skin at the edge of the outer wound, being evidently deprived of life by the impact, was cut away, a couple of further incisions made to expose more fully the lacerated muscles, and many bleeding points tied. The skin was sterilized with carbolic lotion and the wound freely washed with a warm solution of chinosol, the limb being held extended by firm traction on the hand. The torn ends of muscle were brought together with silk stitches, and when the elbow was then bent and the upper arm held straight in that position, the bones were no longer exposed, the wound closing up as to the deep parts and now showing only a portion of muscle exposed in the gap of skin and fascia. Moreover, the oozing of blood was lessened. No attempt was made to close the skin wound, which was purposely left widely gaping to avoid tension and permit drainage. The forearm was kept bent on the arm to less than a right angle and was slung to the neck with a triangular bandage folded and tied round the wrist; and while the broken bone was held in fair line the wounds were wrapped in plenty of sterilized gauze, over which was fixed and bandaged a single splint of sheet iron, so twisted as to lie on the front and outer side of the arm above, passing behind and to the inner side below, some sheets of wood wool tissue being bandaged over all. With the limb thus flexed, slung by the wrist to the neck, lying alongside the body with all this thick dressing around it, and a pillow to rest the tip of the elbow.

the patient lay comfortably in bed with but little disturbance of the bones even when shifting his attitude (fig. 1).

The next day the blood-stained dressings were removed and the twisted splint replaced, thickly padded with fresh gauze, which sufficed as a dressing for the inner wound, supplemented by additional similar splints —a short one in front and an intermediate one on the outer side—all tied together round the arm with strips of bandages. The third dressing was on the fourth day, and the fourth on the eighth day. This time three splints were fixed independently of the large outer wound. A fourth splint the length of the arm was placed over a thick pad of gauze along the outer part of the arm and partly over the other splints above (fig. 2). By this time the discharge was insignificant, and future dressings required only the removal of the outer splint and the gauze on the outer wound. All the injured deep tissues had remained closed in and had coalesced from the first, the discharge being merely superficial (figs. 3 and 4).

During the first week there was a moderate degree of traumatic fever, quickened pulse, irregular bowels and scanty appetite; but in every important respect the patient ran a favourable and painless course and improved daily. He got up on the tenth day and on the eighteenth I left him for twenty-five days, during which the inner three splints were left undisturbed, and with them the inner wound. He went home on the twenty-second day after the injury, coming to hospital twice weekly to have the outer wound dressed by removing the outer splint and changing the gauze.

The series of photographs was taken on the eleventh day, showing the arm slung to the neck with and without splints. A radiograph of the fracture was also taken on this occasion, and had to be done while the iron splints were off (figs. 5 and 6). Although the fracture was as loose as a flail, it kept in line without the splints while the patient sat or stood for the ordinary photograph, but slipped out of place when he was put to lie down for the radiograph (fig. 7). Four days later he was radiographed again without splints, but sitting in a chair, showing good apposition in the absolutely ununited bones (fig. 8). After six weeks the bone was found on my return to be united but not rigid. A radiograph taken that day shows the shadow of callus in front. After seven and a half weeks union was firm and at eight weeks still more so, another radiograph being taken on April 16 (fig. 9).

The points to which I desire to draw attention in this manner of dealing with the fractured humerus are:—

(1) The slinging of the arm to the neck and the flexion of the elbow to a more or less acute angle.

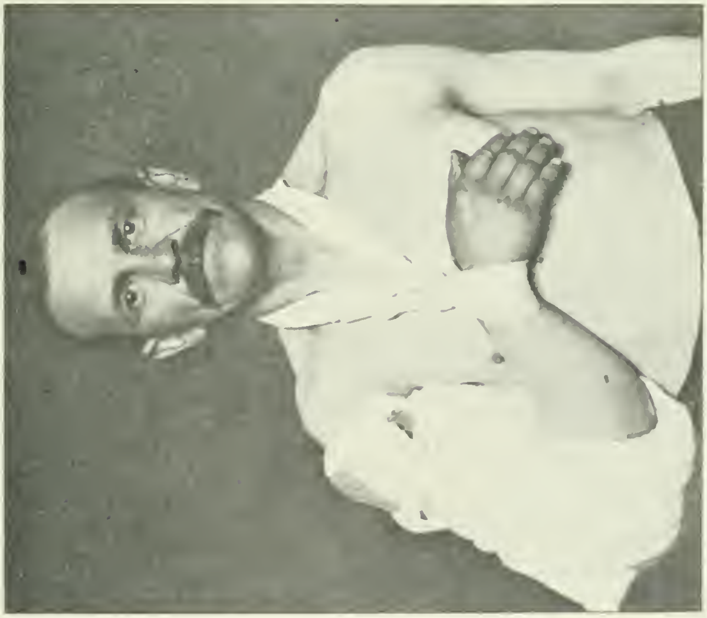

Fig. 1.

Patient in full dressing.

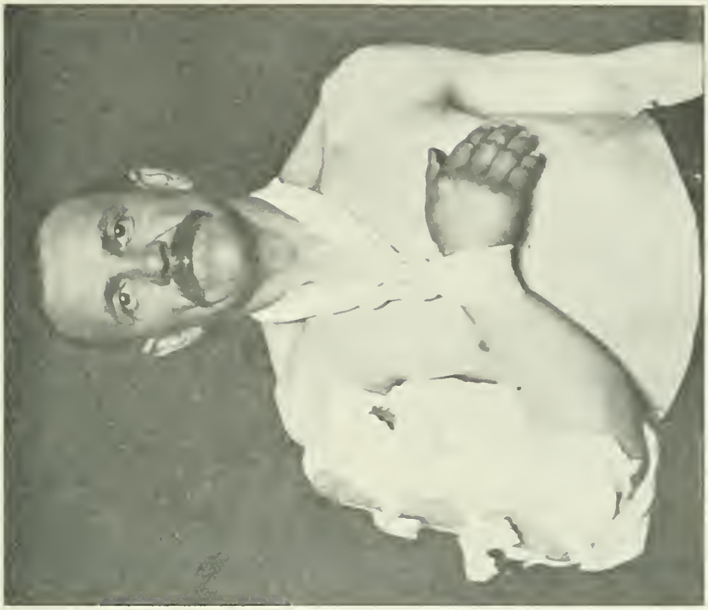

Fig. 2.

Outer dressing mass removed.

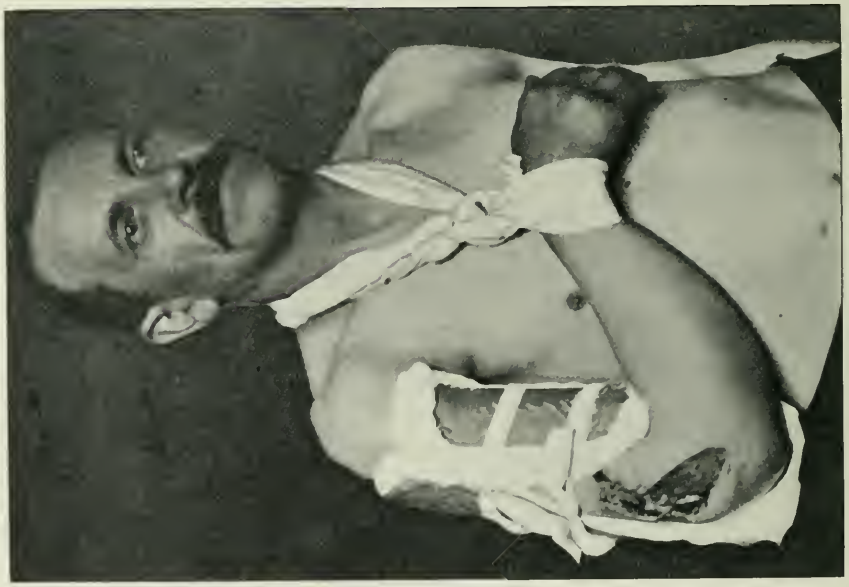

Fig. 3.
All dressing removed except that under remaining splints.

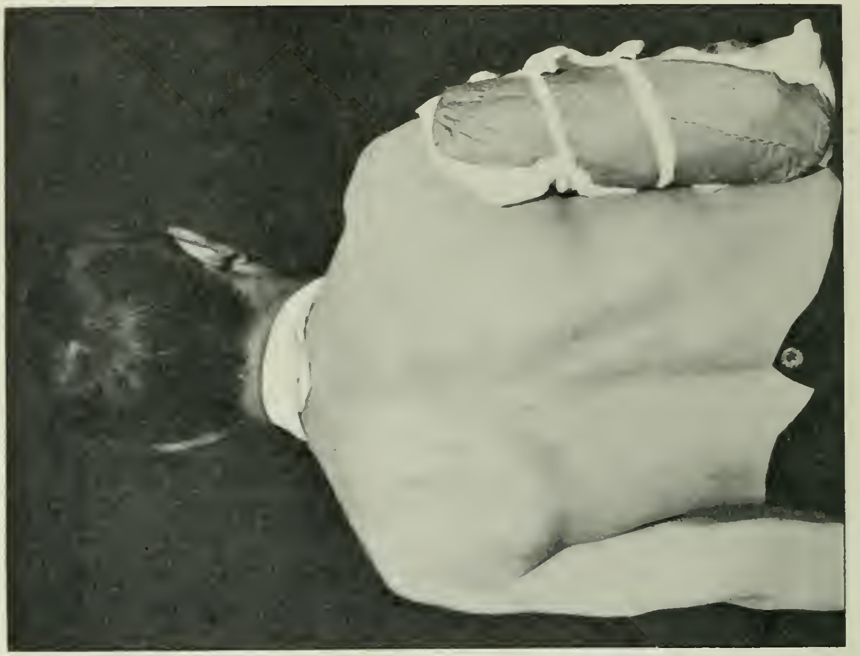

Fig. 4.
Same, back view.

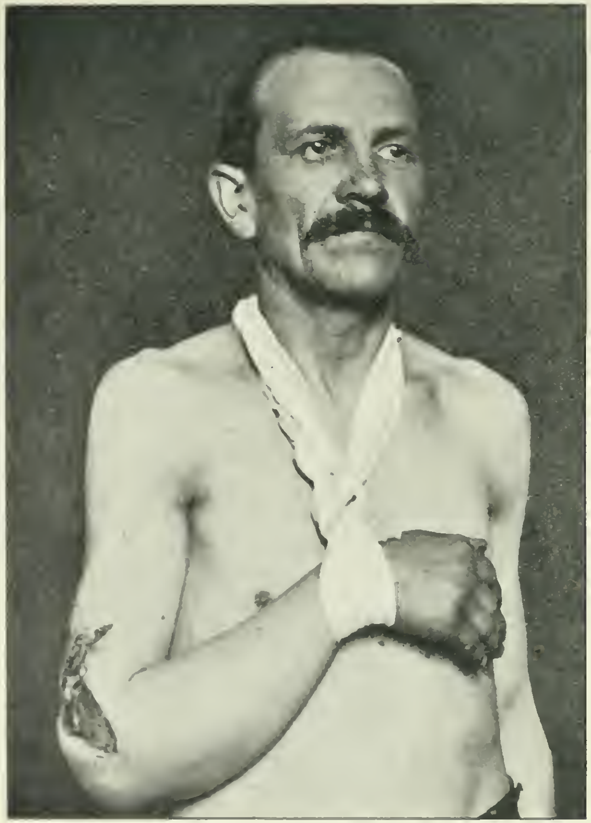

FIG. 5.
Arm slung without splints or dressing. Fracture still as loose as a flail, but not displaced owing to attitude.

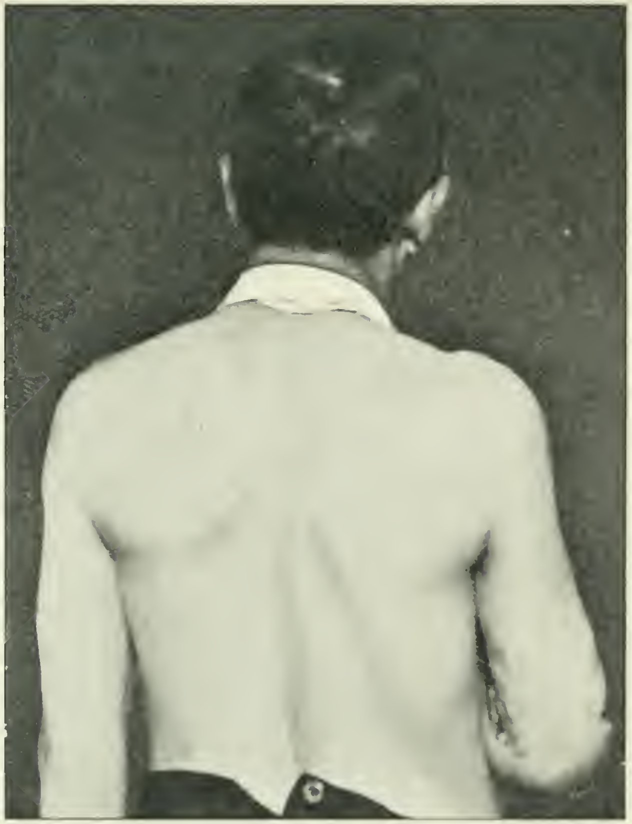

FIG. 6.
Same, back view, showing part of inner wound.

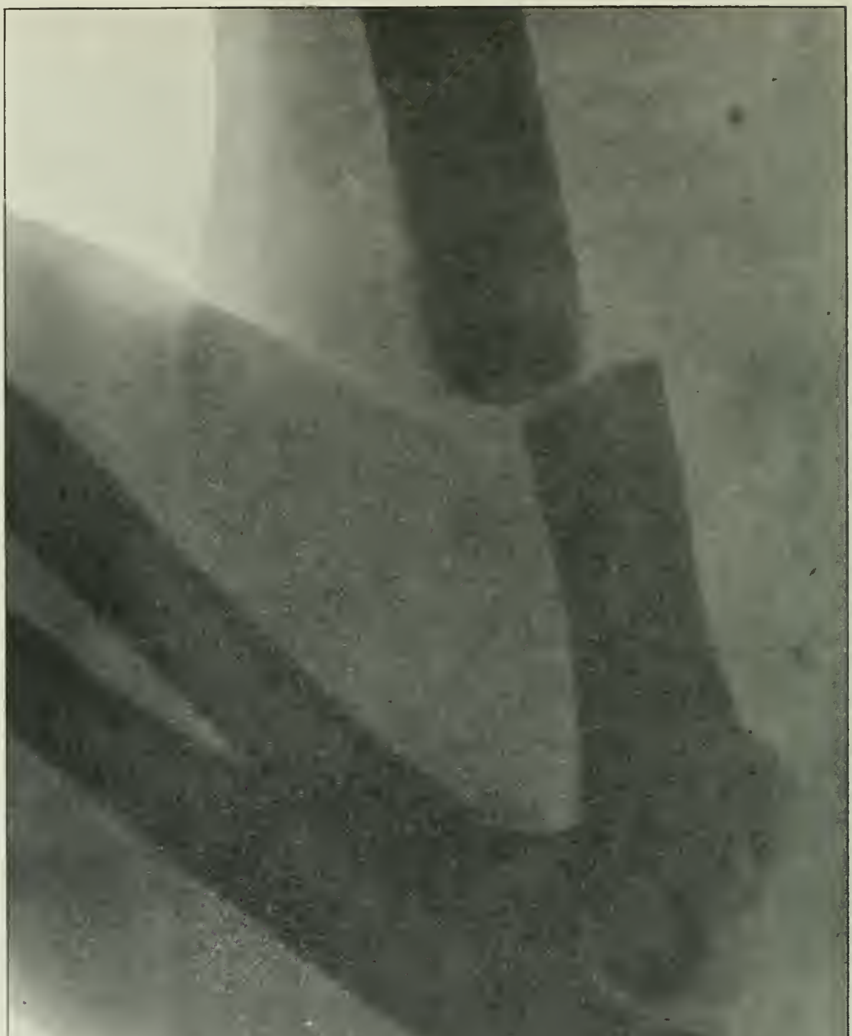

FIG. 7.

Eleventh day after accident; dressings and splints removed. Photograph of patient lying down, in which act the unsupported fracture immediately displaced.

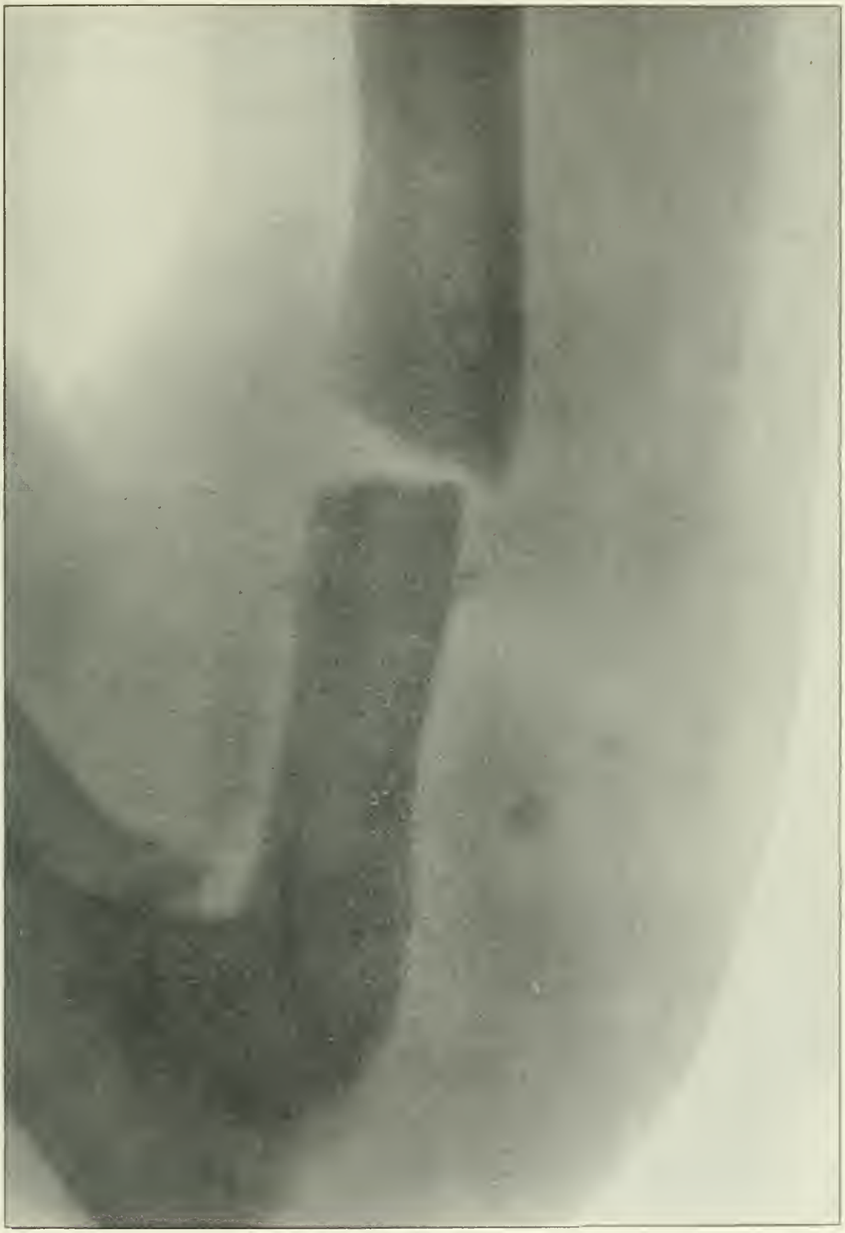

FIG. 8.

Fifteenth day after accident. Same, after removal of splints, but photographed in sitting posture ; fracture not displaced.

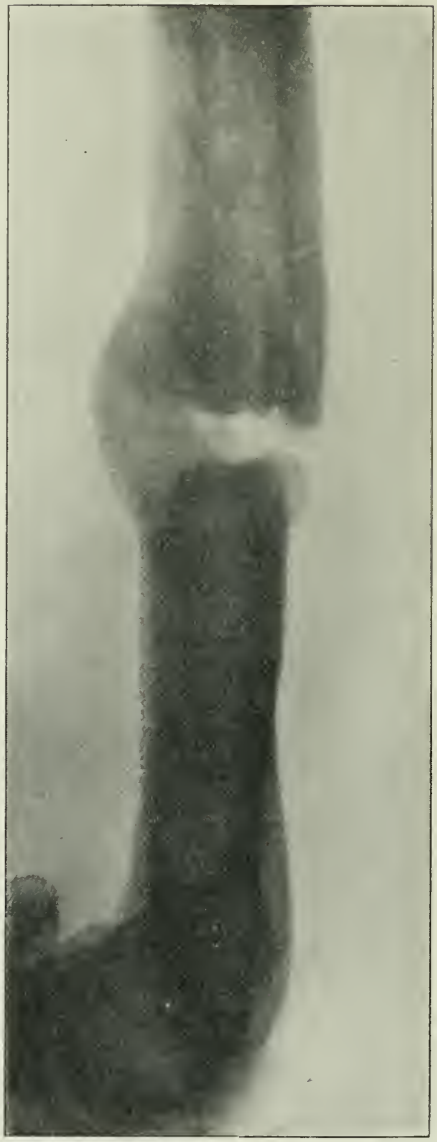

Fig. 9

Photograph eight weeks after accident; callus in front distinct.

The adoption of this attitude in any injury between the clavicle and elbow, both inclusive, is of the greatest service in examination and diagnosis as well as in treatment. By this manœuvre the upper limb is bent into two portions, one of which, containing the hand and forearm, is suspended from the neck; the other portion alone is suspended from the shoulder. In fractures of the humerus the injured upper arm is thus at once relieved of the weight and disturbing inconvenience of the forearm, which, when left hanging loose or not specially provided for, causes pain and deformity to the patient, and ineffectual devices on the part of the surgeon. In fractures of the humerus without displacement this slinging of the disturbing forearm gives so much ease to the patient as to permit a more calm and attentive investigation of the injury. Fractures of the shaft are attended in the loose condition of the limb with displacement and deformity, the precise shape and anatomical explanation of which ceases to be of interest when one reflects that by merely slinging the forearm it does not occur.

In such fractures, when the limb is slung in two halves, the upper arm hangs in line from the shoulder, the muscles are at rest, being no longer called upon to make efforts that in the broken state of the limb are both painful and distorting. This fact was proved to me in 1869 during my house surgeoncy at University College Hospital, though at the time I failed to appreciate its significance. A fellow house surgeon attended for me in the casualty room a case of fractured surgical neck of the humerus, simply slung the patient's wrist to his neck in a piece of bandage, and told him to see me the next day. The upper limb appeared perfectly normal, but on manipulation I found the fracture in the upper fourth of the bone. My astonishment was great; firstly, at what I thought was great carelessness on the part of my colleague, and secondly, because the man seemed none the worse. I thought it incredible that a patient having this fracture could have passed over half a day, in the absence of the usual mechanical support of splints, without pain or deformity. There was the fact, however, which I never forgot, but which some years after became one of the commonplaces of riper experience.

Anyone who saw, and could appreciate, the methods of the late Mr. Hugh Owen Thomas in his management of fractures must have been struck with their masterly character, the directness and simplicity of his control over the injured parts. It was from him that I learnt the adoption of slinging with acute-angled flexion in all fractures of the humerus, and the avoidance thereby of swelling of the fingers, and the tiresome, ungainly formality of bandaging the fingers to prevent that swelling which results in the rectangular attitude of the elbow. The elevation of the hand and wrist well above the level of the elbow itself prevents the hand from swelling, even though the compression of the upper arm in surrounding splints must offer some obstacle to the return circulation through the veins. In the flexed position of the elbow the splints applied round the upper arm must be of different lengths—longer behind and on the outer side, where they may reach the whole length of the humerus; shorter on the inner side, on account of the axilla; and shortest of all in front, because of the diminished room above the bent elbow. Such fractures are more easily treated in the erect position of the patient's body, either sitting or standing; but in severe or complicated accidents the surgeon must manage with the patient lying down. In fractures, also, in the middle of the bone the splints used may be straight, but they should be concave inside to fit the limb better, and, if of wood, are better made of Gooch flexible material. What is far preferable, however, in all cases is the use of flexible sheet metal of some sort, say iron or zinc, ready to hand in many sizes, hollowed to fit the limb and to secure stiffness, lined with a suitable even padding (boiler felt is a suitable and inexpensive material as a basis), covered with waterproofing, or applied bare over the dressings, in case of wound or other source of discharge.

(2) In fracture of the lower fourth of the humerus, on account of the invariable tendency to adduction of the condyles under the weight of the elbow, a twist imparted to one of the splints effectually maintains the lower smaller fragment in line with the upper while controlling both. It is needless to add that this manœuvre is only applicable to a splint of sheet metal. The use of a rectangular splint for the same purpose may, of course, be made to secure this end, but imports into the whole treatment a weight and ungainliness of the limb that no one can tolerate who has learnt to do without it. This in fractures merely of the lower fourth; but every inch higher up in the site of fracture renders the rectangular splint more and more inefficient by tending to lever the lower fragment away from the upper the moment the arm is unslung. The slung arm, with acutely flexed elbow, when fitted with its splints hangs comfortably by the side when the patient is up, and about equally so when he comes to lie down. This attitude of rest not only suits the fractured humerus, in shaft, or either end, but also for treating these joints in other injuries and various forms of inflammation. Even after excision of the elbow-joint I never place the arm to take its chance

more or less extended on a pillow, where every movement disturbs the wound, but prefer to sling the limb, even at a right angle, attached to and supported by the patient's body. This not only prevents all movement, and the pain due to it, but gives so much more freedom to the patient. In fact, in one case of a girl, she begged to be allowed up the day after her elbow was excised, and I was glad to permit it.

(3) In cases of simple fracture it is not difficult, when once the wrist is slung and the elbow bent, and the weight of the forearm removed from the arm, to so apply the splints at the commencement of treatment or by later readjustment as to secure a painless and correct position without hampering the patient's freedom in other respects. But in compound fracture one's first care must be the wound, an aseptic course of which greatly simplifies and shortens the treatment. Even here, in the interests of rest, and for the matter of that of the wound also, the necessary mechanical support need never be totally neglected, and, in the case I have related, was begun in a slight way on the first day and got into respectable form on the second.

I may briefly relate another case, much less formidable, but, like the former, involving the lower fourth of the humerus. I was called in by the late Dr. R. G. Roberts, of Liverpool, to a young man aged 19, who had just had a fall from his bicycle and shattered the right humerus in its lower fourth, with a wound leading to the bone. I took nothing with me except a triangular bandage and several pads of sublimated wood wool, which I laid thickly and pinned around the affected elbow, after slinging the wrist to the neck and suitably cleansing the skin with sublimate solution. By the next day the discharge of blood had ceased, and over the much reduced dressing then required I fixed some sheet iron splints, one of which was twisted so as to keep the elbow and lower fragment in line. The management of the wound was simplicity itself, primary union quickly occurring, and the injury ran a course as favourable as that of simple fracture, and was soundly united and healed in a month, free from deformity and fit for use.

Two Cases of Ununited Fracture treated by planting Small Fragments of Bone between the Ends.

By Rushton Parker, F.R.C.S.

A healthy carter, aged 35, was knocked down on November 23, 1906, fracturing his left humerus about the middle. Five months later the fracture was found not to be properly united. There was evidence of callus and sufficient attachment between the fragments to prevent grating or any movement of the ends past each other, but there was some flexibility and the arm was insufficiently strong for use. When he was admitted into hospital under my care I fancied that union might be secured under a reapplication of splints, bandaged above and below the seat of fracture so as to produce œdema between. The arm seemed to stiffen in a very few days, and then, as the bone was not quite straight, I rectified it by a suitable arrangement of pads on the splints. The bone came straight in two or three days, but its stiffness had become less and the evidence of non-union was greater than ever. Still hoping for union without a cutting operation, I injected iodine liniment with a hypodermic syringe between the fragments on several occasions within a week, but without any benefit (fig. 10). Operation was then decided on and performed on May 30, 1907, by incision in front, planned to avoid the musculo-spiral nerve. The ends of the bones were cleared by scooping and dissecting away a soft unossified medium, and also clipping off a moderate callus on the outer side. The fragments of callus cut small, but of various sizes, were preserved in the blood at the angle of the wound until all was ready to close the opening in the muscles and integuments, when the small fragments were placed between the ununited ends and stitches inserted separately into the muscles and skin. A drainage-tube was put in, the elbow being flexed to less than a right angle by slinging the wrist to the neck; voluminous dressings of sterilized gauze were placed around the upper arm and splints around the dressings. These splints were at first pieces of Gooch's wooden material to permit of X-ray photography through the apparatus, but as the dressings became less voluminous splints of sheet iron, being more adaptable, were used for the more accurate fixation of the bone in line. The wound was healed in about a fortnight; some stiffening at the seat of fracture was perceived at the end of three weeks, and firm consolidation six weeks after operation (figs. 11 and 12).

A lady, aged 48, was sent to me in September, 1907, by Dr. Clemmy, of Bootle, having ununited fracture of the left ulna about its middle. She had fallen on January 27, 1907, and had the break attended to, but being much occupied with invalid parents and other house duties she appears to have neglected her own ailment. There was a bend at the

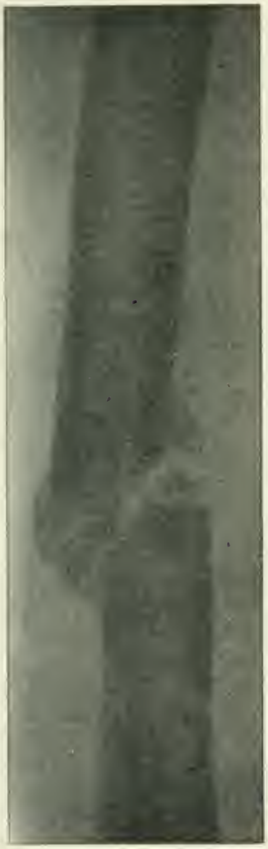

FIG. 10.

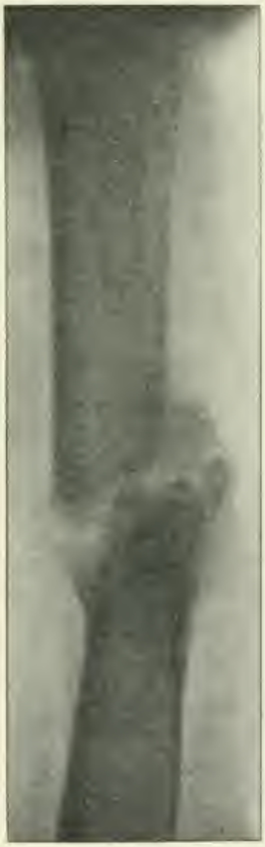

FIG. 11.

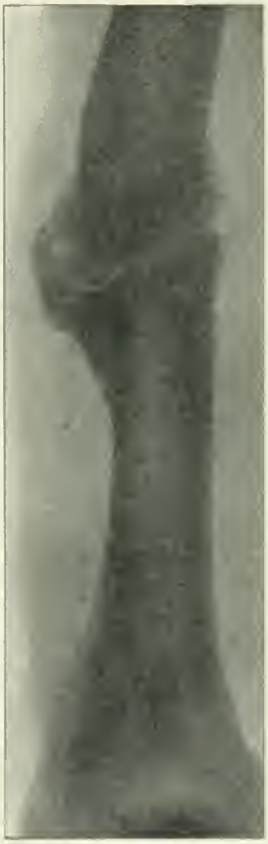

FIG. 12.

Fig. 10.—Two days before operation ; showing callus of flexible imperfectly uniting medium.

Fig. 11.—Six and a half weeks after operation ; union firm, but not demonstrable as such by the shadow.

Fig. 12.—After six months ; shadow of uniting medium as dark as in rest of shaft. Patient lifted a piano the day of this photograph.

seat of fracture, with movement between the fragments on manipulation and a painful state of the affected region (fig. 13). I determined in this case also to attempt to deal with the fracture as in the previous instance, and on September 25, 1907, operated by incision down to the bone, clearing the space between the fragments and planting there such small pieces of living bone as I could collect by clipping bits off the ends, whether redundant or not. These fragments were preserved in warm salt solution during their collection.

The wound was closely sewn, first the muscles and fasciæ and then the integuments, with catgut sulphurized and chromicized (the green catgut of the shops). The sutures of the skin were "buried" beneath the edge to avoid removal. The patient could only arrange for eight days absence from home, after which time she came backwards and forwards to see me. She only kept her bed a couple of days, and healing of the wound was complete within a fortnight.

At the first dressing, at the operation, the wound being the chief care on this occasion, no attempt was made to be particular about the exact position of the broken bone, though splints were put on over a voluminous gauze dressing folded so as to serve as a pad around the whole forearm. But the day after, and on all subsequent occasions, the bone was well pulled into line and the splints carefully arranged to keep it so. Here also two pieces of Gooch's material were used, and X-ray photographs taken through it without disturbing either splints or dressing, or impairing the radiograph.

These records were taken the day after operation and weekly afterwards, in addition to those before operation. At the end of three weeks a single splint sufficed. By the end of four weeks there was distinct stiffening, but a week later the fracture was still ununited, so the splint was removed, the forearm allowed to hang down, and massage night and morning advised so as to encourage a little swelling and improve the nutrition of the region. A few days later there was more stiffening, but at the end of six weeks there was still no firm union. I then gave the seat of fracture many blows with a small hammer, a piece of leather being interposed, and this the patient endured without anæsthetic. In some of the final blows I found that I had torn open the upper end of the scar, which was then covered up in some carbolized cyanide gauze and bandaged, and the limb still permitted to hang down and be used lightly. A couple of days later this opening had closed again, and by the end of a week—that is, seven weeks after operation—the fracture seemed firmly united and has continued rigid ever since (fig. 14).

The practice followed in these two cases is based upon the experiences of Sir William Macewen, which were published in the *Proceedings of the Royal Society* for June, 1881. The subject was again brought before the notice of the profession in December, 1906, by the same distinguished surgeon in his lecture on the regeneration of bone at the University of Liverpool, on which occasion my own attention was directed to it more fully and accurately than before. I had previously attempted bone-grafting, but the fragments used were too large, and the

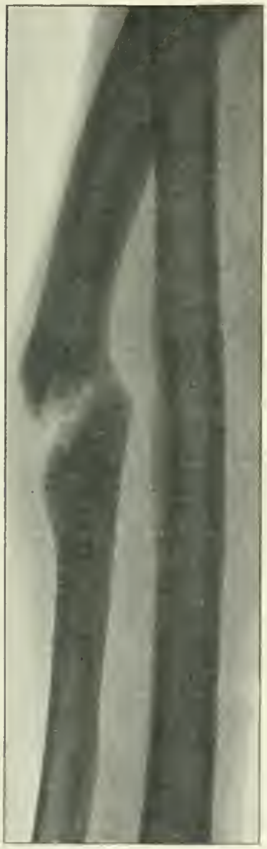

FIG. 13.

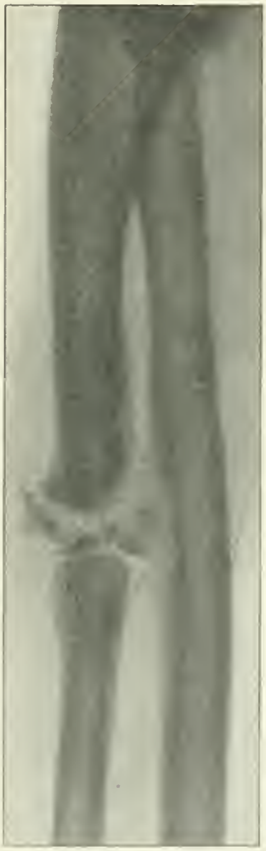

FIG. 14.

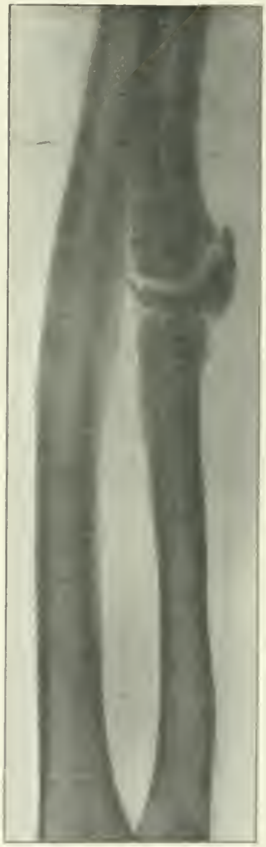

FIG. 15.

Fig. 13.—Before operation, same day.

Fig. 14.—Seven weeks after operation; union fairly firm, but shadow chiefly in planted fragments.

Fig. 15.—Five and a half months after operation; showing coalesced fragments in dark uniting medium.

attempts failed. Sir William Macewen again brought the subject before the Royal Society in January, 1907, an abstract of the paper being published in the *Proceedings* of June and in the *British Medical Journal* of June 22, 1907. It may be stated quite briefly as follows:—

(*a*) Bone is developed from periosteum when bone cells are already adhering to it. Periosteum without adhering fragments of bone does not develop bone. The amount of callus depends on the limitation of the periosteum and the amount of movement.

(*b*) Bone deprived of its periosteum continues to grow. Bone may be made to grow in the midst of lacerated muscles by the mechanical distribution of osteoblasts; and shavings of nude (denuded, bare, or naked) bone grow on being placed between muscles in a gap in the continuity of the shaft.

The cases of ununited fracture, so called, are at times devoid of all attempt at bony attachment between the fragments, the limb being almost as loose as a flail, but more often there are masses of callus, showing efforts at union; in fact, a condition of not exactly "ununited" but rather of "imperfectly united" fracture, as in my first case here related. If in such cases the unossified medium be cleared away and an ossifying medium substituted, much of Nature's work can be preserved and the rest completed by art, in fact the whole operative procedure may undoubtedly be simplified, and I venture to recommend it to the attention and practice of other surgeons. The operation of wiring, often considered the only or the main resource in ununited fracture, is successful enough to be a recognized operation in surgery; but it is beyond all comparison more difficult, and more disturbing to the limb, than the proceeding here described. On the comparatively small scale of the forearm, or that somewhat larger of the upper arm, the wiring method may be devoid of much severity, whereas in the thigh it may be really formidable to the surgeon, dangerous to the patient, even when skilfully carried out, and not unfrequently fatal from hæmorrhage and other complications. The statistics of ninety-eight published cases in 1881 give nineteen deaths, which is nearly 20 per cent.

Dressings.—As in all compound fractures, the first consideration is the wound, which at the first dressing should be voluminous, to soak up the free early discharge of blood. On this occasion the exact arrangement of splints is of secondary importance, and when not convenient or easy may even be left in abeyance; but in a day or two, when the discharge has lessened, the mechanical fixation of the limb can be secured.

Palliative Measures.—The improved adaptation of splints, when followed by the benefit of union, can only have been of use at a comparatively early date after the fracture, and so during "delayed union." It can only be classified with the primary application of splints, of which it is a continuation. Another method, rubbing the fragments together, and the administration of whisky, as practised by the late Sir William Fergusson, is spoken of as having succeeded in delayed union. The object to be attained is the production of irritation, whereby the tendency to bone formation, that has fallen into abeyance, may be increased. The uncertainty of this device is too obvious to merit serious discussion, especially when, as often happens, the fragments cannot be rubbed together. But on pursuing the principle—that of setting up a fresh bone-forming irritation, an osteoplastic ostitis, or even periostitis—it is possible, by more rational and thorough means, to attain the object. Bandaging above and below, so as to congest the seat of fracture, will sometimes attain this end, and I have succeeded with it; but success is only possible in delayed, not in actual non-union, and it failed in Case I. This device, and the more thorough procedure of hammering, were freely practised by the late Mr. Hugh Owen Thomas, from whom I learnt them. Cases of delayed union can generally be easily made to unite by hammering the region of fracture, and I have succeeded in every case to which I have seriously applied it. The second case recorded this evening is a case in point. After bone-grafting, the fracture was still in a state of delayed union at the end of six weeks, and it is of course possible, though it did not seem likely, that union was eventually to occur. However, a week after the hammering the union was firm. In Case I. both the congesting method and that by injecting iodine liniment as an irritant were carried out, but without success. A beginning was also made with a slight hammering, but this was practised without anæsthetic in a tender locality, and was in no way thorough or efficient. Of the method by drilling and inserting pegs of bone or ivory, designed for the same end—that of setting up a bone-forming irritation—it is hardly possible to speak, with the slight experience I have had. In the only case in which I ever did it no irritation whatever was set up, and I have never tried to add to my experience in that direction, being unable to see any grounds for belief in its use.

For the great service rendered in the X-ray department by Mr. Thurstan Holland and his assistant, Mr. Woods, I wish to express full acknowledgment. Many things have been learnt by the revelations of this interesting application of science; among others, the fact that up to two and a half months after union, procured by bone-grafting, the shadow around the grafts continues so attenuated as in nowise to demonstrate the fact of union. In examining the shadows in figs. 10 and 11 it would be impossible to decide between the flexible imperfectly uniting medium (fig. 10) and the firm union (fig. 11). It was not till after six months that, in the first case, the shadow at the seat of union became as dark as that of the unbroken shaft (fig. 12). In the second case I have had the bone radiographed at intervals, and on the last occasion, five and a half months after operation, a deep shadow appeared in the uniting medium developed from the grafts (fig. 15).

PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

VOLUME THE FIRST

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SESSION 1907-8

HERAPEUTICAL AND PHARMACOLOGICAL SECTION

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THERAPEUTICAL AND PHARMACOLOGICAL SECTION.

CONTENTS.

October 22, 1907.

PAGE
Presidential Address. By T. E. Burton Brown, M.D. 1

Discussion on "The Treatment of Functional Dyspepsia." Opened by Robert Hutchison, M.D. 3
Dr. Habershon (p. 7). Dr. Herschell (p. 8). Dr. H. C. Cameron (p. 10). Dr. J. Gray Duncanson (p. 11). Dr. Hutchison (p. 12).

November 26, 1907.

The Therapeutics of Indigestion. By William Murray, M.D. 13

Some Tropical Diseases and the Remedies required for their Treatment and Prophylaxis. By James Cantlie, F.R.C.S. 22

December 17, 1907.

The Action of Digitalis on the Human Heart. By James Mackenzie, M.D. ... 29

Reminiscences of an Apprentice Fifty Years Ago. By William Soper 32

January 28, 1908.

PAGE
Arteriosclerosis. By W. E. Dixon, M.D. 33
Nutmeg Poisoning. By A. R. Cushny, M.D., F.R.S. 39

February 25, 1908.

The Therapeutics of Diet. By Harry Campbell, M.D. 45

March 24, 1908.

Hypnotism. By J. Milne Bramwell, M.D. 49

April 28, 1908.

Remarks on Rheumatism and Chorea in Childhood. By Leonard G. Guthrie, M.D. 65

The Council think it right to state that the Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Therapeutical and Pharmacological Section.

October 22, 1907.

Dr. T. E. Burton Brown, President of the Section, in the Chair.

PRESIDENTIAL ADDRESS.

Gentlemen,—It is now my duty to open the first session of the Therapeutical and Pharmacological Section of the Royal Society of Medicine, and I would wish at the same time to give a brief account of the Therapeutical Society, from which our Section was derived, and of which I have every reason to hope it will be a most flourishing successor.

The Therapeutical Society was commenced in July, 1902, after a suggestion made by Dr. Robert Lee, who is now a Vice-President of this Section. A meeting was held in the Apothecaries' Hall, and it was unanimously agreed that a Therapeutical Society should be formed in connection with the Apothecaries' Society, and the Master of the Company induced the Court of Assistants to allow the new Society the use of this venerable hall, with its furniture, firing and lighting, without any charge. This kindness has been of the greatest value to the Society, enabling it to work without any deficit as long as this permission continued. On August 6, 1902, the first meeting of a pro-council took place, and on January 20, 1903, the first paper was read to the Society by Dr. Gordon Sharp on *Alstonia*. Our first President, Sir William Thiselton-Dyer, gave the Society the greatest assistance by presiding with the utmost skill and urbanity at our meetings, and especially by inducing several scientific gentlemen to read valuable papers at the meetings of the Society. The number of the Fellows of the Society greatly increased while Sir William Thiselton-Dyer remained President. He was succeeded by Sir Lauder Brunton, who very greatly promoted the therapeutical work, not only by his assiduous attendance at all the general meetings and committees of the Society and his cordial kindness, but also by his thorough acquaintance with every topic brought before its members and the valuable information which he gave in every discussion. Under his presidency the Society continued to improve, not only in the number of Fellows joining it, but in the extent of the papers read, so that while the first Transactions only comprised 35 pages, the last series, which has just appeared, contains 176 pages, having been most ably arranged by our late Secretary, Dr. Herbert French. Among the valuable papers published in the past five volumes of our Transactions are some of great importance from Dr. Burnet, Dr. Dixon, Dr. H. French, Dr. R. Hutchison, Mr. Maiden, of Australia, Dr. G. Sharp, Dr. Nestor Tirard and Dr. Wild, all Fellows of the late Society, besides others of no less importance from gentlemen not connected with us, namely, from Professors Cash, of Aberdeen, Koberl, of Rostock, and many others.

In 1905 a proposal was made that most of the medical societies in London should unite to form the Royal Society of Medicine, and have their head-quarters at 20, Hanover Square. This project was under discussion for some time, and the Secretaries of the Society were appointed delegates to ascertain the terms on which we could be admitted to the Royal Society. It was found that it was not possible to maintain the low rate of subscription of half-a-guinea or five shillings, which had been paid heretofore, but that it must be increased to one guinea from every one who wished to be a member of the Therapeutical Section only, and to three guineas from those who desired to become Fellows of the Society with the right to attend the meetings of every Section and the privilege of using the library, eight books being allowed to be taken out at a time by any Fellow, while members have a right to attend only the meetings of the Sections to which they belong, but to receive all the papers published by the Society in any Section. No one can vote on any business matter in a Section unless he is a member of that Section. Members can have the use of the library by the extra payment of one guinea. These are valuable privileges, and it is to be hoped that many of the Fellows of the Therapeutical Society will join the Royal Society of Medicine.

When this arrangement was put to the vote of the Fellows of the late Therapeutical Society, only fourteen were unwilling to join the new Society, and in consequence of our last July meeting it was agreed that the Therapeutical Society should cease to exist, and that it should be converted into the Therapeutical and Pharmacological Section of the Royal Society of Medicine, and as such may it long survive and prosper. We have every reason to expect that it will do so, especially under the assiduous attention and careful working of our present Secretaries, Dr. Gray Duncanson and Dr. Cameron; and we anticipate that we shall be able in our new position to do much to promote the extension of therapeutical and pharmacological knowledge in the future, subjects which are of the greatest importance in the treatment of disease, and which, I fear, have been somewhat neglected of late.

A Discussion on the Treatment of Functional Dyspepsia.

Dr. Robert Hutchison, in introducing the discussion, said the first essential was to be quite sure about the terms employed, because a fruitful discussion could not take place unless it was certain that the members were dealing with the same thing. By dyspepsia he meant some discomfort or pain, an abnormal sensation of some kind, experienced during the process of digestion. He would exclude abdominal pains which had not a definite relation to the process of digestion. Dyspepsia might be due to organic disease or functional disease of the stomach. The organic cases, the causes of which were cancer of the stomach, ulcer and gastritis, he did not propose to deal with, because there was a general agreement with regard to their treatment. It was true of medicine as a whole that, in a disease with a definite pathology, the treatment was fairly definite; it was where we had to deal with functional disorders that confused ideas and methods prevailed. In regard to functional dyspepsia, the first essential was an accurate classification. He did not think progress in dealing with the treatment of functional disorders, either practically at the bedside or in discussions, would be made until a more accurate classification was obtained than had hitherto existed. The cases under discussion were usually spoken of loosely as "acid," "atonic," "flatulent," &c., dyspepsia; in fact, all kinds of names were used. It was necessary to have a more rational or scientific classification, and he had sketched out one which he believed to be useful, based upon the physiological functions of the stomach. The physiological functions of the stomach were (1) secretory, (2) motor, and (3) sensory. The stomach had no other function except that of absorption, which was not important from the present point of view. Any cases of functional dyspepsia must be due to disorder of one or other, or several, of those physiological functions, either in the direction of excess or defect. In the case of the secretory function excess was represented by *hyperchlorhydria* and *hypersecretion*, and defect by *hypochlorhydria* and *achylia*. In the case of the motor functions, excess was represented by *pyloric spasm* and "*tormina ventriculi*," and defect by so-called *motor insufficiency*. In the sensory function excess was represented by *hyperæsthesia* and *gastralgia*. As to whether there was any defect or deficiency of sensation responsible for cases of dyspepsia it was impossible to say. He thought it was conceivable that some cases of profound loss of appetite might be due to some deficiency or minus degree of sensation. We did not, however, know enough about those cases to discuss them profitably. All the ordinary clinical types of dyspepsia were due to combinations of the disorders already described. A very common combination was an excess of acid along with an excess of *motility*, which often went on to the production of pyloric spasm. There were also such combinations as a defect of acid along with a defect of motility. If every case of functional dyspepsia could be analysed, it could be separated up into a combination of one or more of those different functional disorders. He admitted that the most rational way of dealing with dyspepsia would be to go further back still, as he agreed that the majority of these functional cases were due to some disorder of the nervous system, and therefore the most rational way of dealing with dyspepsia would be to go further back and put the nervous system right first, to calm it down, or to strengthen it and make good the loss of nerve energy. That could not always be done in practice owing to the circumstances of the patient's life, and therefore it was necessary to deal with the more immediate conditions existing in the stomach itself. All that one could do in the majority of cases was to deal with the functional disorder directly, so as to relieve the patient of his discomfort. Under the head of Treatment he had put down, in the first place, some general considerations applicable to all cases, which he did not propose to deal with in detail, because they were points about which every one was agreed. It was unnecessary to press the importance to dyspeptics of such things as a due proportion of rest, exercise, and fresh air. A factor which could not be exaggerated was the importance of avoiding chill. Very frequently dyspeptics found that a chill put them wrong more easily than indiscretions of diet. It was also important to avoid pressure upon the stomach, and to pay attention to the teeth and the proper regulation of meals. He did not know that there would be so much agreement as to the use of tobacco and alcohol, but that was such a large subject that he could not enter into it on the present occasion, nor on the kindred subject of tea and coffee, and the part they played in producing functional disorder. He also did not propose to deal with the use of aperients and hydro-therapeutic methods. What he desired to consider was the methods at hand for putting right the disorders of function to which he had referred. In that connection he wished to deal first with the treatment of *excessive secretion*. The only rational way was to diminish the secretion if possible, and the question was how that should be done. He thought it was important in the discussion to keep separate the use of diet and the use of drugs. Both had their place; both could be used to remedy any defect of function, but it was important to discriminate between them. Personally, he thought the point that diet played in the production of dyspepsia was greatly exaggerated. It was a gross libel on dyspeptics to say that dyspepsia was usually the result of over-eating and over-drinking. He thought it would be far nearer the truth to say that dyspepsia was the result of over-work, and exhaustion of the nervous system consequent upon it. To return to the case of excessive secretion, one should avoid any articles of diet which stimulated secretion, such as extract of meat, spices and condiments. It was more important, however, to use diet as a means of neutralising the acid once it was present. He suggested that cases of excessive secretion were best dealt with, not by trying to stop the secretion, but by frankly recognising that the excessive secretion was there and neutralising it by an arrangement of the diet. He knew that some people took another view, and gave foods which did not cause so much acid to be secreted, but he thought experience showed that that line of practice was not successful. If the acid was neutralised it did not leave free acid present in the stomach to the same extent, and it was the latter which mainly caused irritation. He suggested that in those cases the *food should be used as an antacid*, just as one used alkaline drugs, and the best foods for the purpose were those rich in proteid, *i.e.*, animal foods. Drugs might also be rationally used to diminish or neutralise secretion. But his experience of drugs, such as belladonna, bromides, nitrate of silver and astringents, was that they did not much diminish the amount of acid in the stomach. He was not sure that bromides did not; at all events, he knew that better results were sometimes obtained by combining bismuth with bromide than by using bismuth alone. That was the only drug which he felt sure was useful in diminishing the secretion. On the whole, he thought it was better to content oneself with neutralising the secretion, and there was a large selection of alkaline remedies for the purpose, such as the soluble and earthy carbonates. It was best to use the earthy carbonates, the only disadvantage being that they produced an excessive ebullition of gas, which was sometimes unpleasant. The line of treatment in *diminished secretion* was diametrically the opposite. For instance, one would use as a diet foods which stimulated secretion, but he did not think there was any advantage in accommodating the diet *chemically* to the impaired secretion. So long as the motor power of the stomach was unimpaired, it did not seem to matter if there was no secretion. The important thing, therefore, was to see that the food was so prepared that it would easily leave the stomach. As regards the use of drugs, there were plenty which stimulated secretion, particularly bitters. Alkalies used to be given before meals in small doses to stimulate secretion, and he desired to know the opinion of the members as to whether they were useful or not. He was not sure that small doses of bicarbonate of soda did not increase secretion, although this was opposed to scientific teaching. With regard to replacing secretion, he thought it was hardly ever necessary to give hydrochloric acid and ferments: he believed that was bad therapeutics. He distrusted the man who was always prescribing pepsin and such ferments; he believed most of them were inert, and, even if they were active, were unnecessary. As long as the motor power of the stomach was unimpaired, the pancreas could be left to carry on digestion. He was not quite so sure about the hydrochloric acid, because it was useful in some cases in stopping gastric diarrhœa. It was possible that the hydrochloric acid there acted as an antiseptic and not as a digestive agent. He had made up his mind that the ferments were almost of no use at all. With regard to the treatment of *motor spasm*, it was associated with excess of sensibility, and very often with excess of secretion. The treatment of the motor excess resolved itself into treating the excess of acid or sensibility which were indirectly responsible for it. The best method of dealing with it was the local application of heat; there was nothing so good for pyloric spasm as assiduous poulticing, which had rather fallen into disuse. The treatment of *motor insufficiency* was one of the main points on which he wished to speak. Was there such a thing as motor insufficiency at all? He meant by it a condition in which the stomach did not empty itself quickly enough, a condition which was often associated with splashing over the stomach, although he did not say that every person who had splashing of the stomach had motor insufficiency. Assuming that such a condition existed, what were the agents at their command for improving the motor power of the stomach? That was, he thought, the weakest part in their treatment of functional dyspepsia. Diet could not be used to increase motor power directly. If one was treating a lame stomach, the food must be so prepared that it would leave the stomach with the least possible trouble. A dry diet was of great use in such cases, because such stomachs could not stand fluid, being over-distended by the sheer pressure of the weight of the fluid. Were there any drugs which stimulated the motor power of the stomach in the same way as digitalis stimulated the heart or ergot the uterus? Strychnine generally did a patient good in such cases, but he doubted if it increased the muscular power of the stomach. Digitalis might probably act on the stomach. Ipecacuanha used to be given as a gastric stimulant, but personally he did not believe there was a drug to be depended on to make the stomach contract better. Were physical methods of any use in such cases? For instance, was massage useful in making the stomach contract? He had known patients improved in that way, but he thought possibly it was because their general condition had benefited. He was not at all clear about the use of electricity, but if there was any type of dyspepsia in which electricity was likely to be beneficial it was in motor insufficiency, and undoubtedly the sinusoidal current was the best form to use. The treatment of *hyperæsthesia* was very much like the treatment of over-acidity, with which it was so often associated. It was important to have an unirritating diet, and it was sometimes necessary to use diet to improve the patient's general nutrition; one had to stuff the patient until he got well and strong. Fortunately, a whole host of drugs could be relied upon in those cases. Personally, he believed, there was none so suitable as bismuth, although how it acted he did not know. Bismuth freely given was suitable in almost all forms of dyspepsia. In the last place, it was important to recognise the supervention of an element of gastritis upon functional dyspepsia; but everyone was agreed as to the treatment of gastritis if it occurred. In conclusion, he desired to plead for a more scientific classification, in order to make not only the diagnosis but the treatment of functional dyspepsia more satisfactory. What he thought they most needed to discuss was (1) whether such a classification was possible, and to what extent it was capable of being used at the bedside; and (2) what were the best methods of dealing with the atonic type of case.

Dr. HABERSHON remarked that in olden days dyspepsia used to be classified according to the symptoms, but it was not possible to do that nowadays because it was found that many of the symptoms occurred in almost every branch of the disorder. A truly scientific classification, such as Dr. Hutchison had given, might be most helpful in the treatment of the disease. He thought it must be remembered that in dyspepsia there were many causes which assisted in the development of certain symptoms. In the treatment of all dyspepsias, he thought the first thing was to find the true scientific cause of the disease. When that was done half the battle was over. In dealing with the symptoms that arose from the disease, not only the therapeutical use of the drug had to be considered, but also the peculiarities of the patient. The whole question of drug treatment had been enlightened by the wonderful researches of Professor Pawlow, and he believed there was still a great field for experimental therapeutics. At the same time that was only the physiologist's point of view, while the physician had to deal with the idiosyncrasies of the patient, which necessarily had to do with the character of the drug and the dose. He cordially agreed with Dr. Hutchison in most of his remarks, especially with regard to the great difficulties in the treatment of motor insufficiency of the stomach. He thought all medical men would acknowledge that the cases of dyspepsia which baffled them most were those of central nervous origin, the neurotic patient with dilatation of the stomach. The first thing to do was to help the stomach to empty itself, and all the mechanical means mentioned were of great value for enabling that to be done. The empty stomach lay in a semi-contracted condition, and although there might be deficient muscular power, it was more likely to return to that condition when empty than when full. He believed in massage because it emptied the stomach, but he had not much faith in electricity, most of his cases which had been dealt with in that way not gaining much benefit. He was afraid also that he knew of no drug which would do good except by dealing with the cause. If the cause was of central origin in a nervous patient, strychnine and phosphorus were sometimes of much assistance. With regard to the use of bicarbonate of soda, he thought Professor Pawlow went too far when he asserted that it did not increase secretion. He was quite sure that large doses checked secretion, but small doses given before food excited secretion, whatever the physiologists might say. He cordially agreed with Dr. Hutchison's remarks about the so-called zymine ferment. It never reached the place where it was wanted; it was destroyed by the acid of the stomach, or, if it passed through, it reached the intestines before the pancreatic secretion was exerted. He thought there were some cases where pepsin added to hydrochloric acid was of great value. Personally he would like to see pure gastric juice drawn from an animal, from a fistula, because he believed that would be of great use in cases of secretory insufficiency.

Dr. HERSCHELL thought it was possible to go a little further than the author had gone in the classification of functional derangements of the stomach. He was in the habit of adopting the following classification of cases: In the first class he put cases in which the functions of the stomach were normal when they were tested, *i.e.*, the motility and the secretions were normal, and the patient suffered discomfort from digestion only when an increase in the amount of food was taken. That was the mildest class of disorder. The second class was where the functions of the stomach were normal, but it was unable to deal with a normal meal without producing some discomfort. In the next place there were alterations in the secretion and in the motility, which he agreed with Dr. Hutchison formed distinct classes of investigation; and there was a fifth class in which there was a combination of the alterations in secretion and motility, excessive and diminished secretions being obtained. It was all very well to form a method of treatment based upon that classification, but it was necessary to find out what the patient was suffering from, and he was sorry to say in most cases of dyspepsia the physician did not attempt to do anything of the kind. He maintained that so many functional disorders might be due to serious organic disease that it was the doctor's duty in every case, excepting a very slight one, to make a thorough investigation of the conditions of the secretion and motility of the stomach, and find out what happened to the food after it went into the patient. In a case of slight duration, which was not very severe, one might be content with the method of trial and error. After some practice it would be possible to deduce from the symptoms of the case pretty nearly what was the matter; medicine was given, and the correctness of the diagnosis proved or disproved by its effect. But in severe cases thorough investigation was necessary. A very great deal could be done by simply examining the empty stomach. If a little of what there was in the stomach was extracted before breakfast by a tube with an indiarubber bulb, and a microscopical examination made, a diagnosis could nearly always be arrived at. He had very little to say with regard to treatment after the excellent suggestions made by the author. In the treatment of excessive secretion it was necessary to wash the stomach out. The patient should be put in bed and treated like a case of ulcer of the stomach. For the first two days nothing was given by the mouth: a period of milk diet followed, and the diet was gradually increased until it was normal. If the patient was unable to take milk, lime water should be added to it, and he should be given a sedative in order to be able to retain the milk. One drug which the author had not mentioned, which appeared to have considerable effect in reducing secretion, was ergot.

Reference had been made to the use of carbonate of soda. The reason why the earthy carbonates should be used in preference to bicarbonate of soda was twofold. First of all, bicarbonate of soda was very soluble, so that if it was taken into the stomach it at once dissolved, saturated the system, and the blood was made alkaline to too great an extent; whereas if carbonate of lime was used only sufficient was dissolved to neutralise the acid. It was better in such cases to use magnesia, because, by regulating its amount, the constipation which so often took place could be obviated. Carbonate of soda in small doses, when given before a meal, increased the secretion of hydrochloric acid, but hydrochloric acid in the treatment of deficient secretion had always failed in his experience. He had, however, obtained good results from the use of pepsin. Dr. Hutchison had said he had never seen any good results from the use of electricity in the treatment of motor insufficiency. The reason why that was frequently the case was because its application was too often left in the hands of unqualified people—for instance, nurses whose knowledge of electricity consisted of four or five weeks' instruction in a massage school. Naturally the patient received no benefit, and the doctor said the electricity had failed. As a matter of fact, electricity properly applied was one of the very few stimulants which it was known would act upon involuntary muscular fibre, and the current which was best was the sinusoidal. Personally he always used the tri-phase current, because there were three electrodes, each of which became positive in turn and the others negative, so that if they were applied to three parts of the body there was a zone of the body being permeated by the current in different directions. It was his experience that patients obtained a good deal of benefit from the use of electricity when used by experts. The fact that experimentally electricity would not contract the stomach was no argument against its use in atony, as in these cases there was very rarely any actual weakness of the muscular fibres, the motor defect being due to defective innervation, and the effect of electricity in toning up the nervous plexuses governing the stomach being undeniable.

Dr. H. C. Cameron remarked that he had been working in the Physiological Laboratory for some time on problems which were not very far removed from the subject of discussion, viz., the digestion of patients after the operation for gastro-enterotomy. The author's remark that bismuth in large doses could not do harm was perfectly true. He, personally, had been taking two ounces of carbonate of bismuth, and giving it both to patients and friends, in order to photograph the stomach on the X-ray screen. In doing the work he had been struck with one fact with regard to the treatment of an atonic stomach. One would imagine that in treating this condition a food should be given which required as little movement of the stomach as possible to pass it onward, and he had noticed that for this purpose milk was about as bad a food as it was possible to give. After a meal of milk, the bismuth shadow persisted quite as long as after an ordinary meal. If the doctor wished to treat an atonic dyspepsia, he should take steps to prevent coagulation and to preserve the fluid condition. From Pawlow's work he should have said that in treating cases of hyperchlorhydria success would be obtained by giving a diet extremely rich in fats. Pawlow had shown conclusively that fats were the part of the diet which most of all diminished gastric juice; and a diet rich in fat, given at frequent intervals, would undoubtedly abolish, in a case of gastric ulcer, the excessive secretion of acid. A diet rich in fat, together with large doses of alkalies, diminished hyperchlorhydria, and was the method of treatment indicated.

Dr. J. GRAY DUNCANSON stated he had been pleased to hear in the course of the discussion that ferments had been found of little service in the treatment of dyspepsia. As a general practitioner he had practically given up the use of pepsin. In neurotic dyspepsia, a pill formed of phosphorus, strychnine, and iron was an excellent remedy. There were two forms of motor insufficiency of a temporary nature which were most interesting to study. The first was the motor insufficiency and diminished secretion seen in sea-sickness. The stomach, having emptied itself, was left in an absolutely atonic condition, and there was also a distinct diminution of secretion, with a reluctance to eat. That form of trouble arose from a central origin; it might almost be called giddiness of the stomach in many cases. He was not referring to people who over-fed themselves beforehand, but to people who had taken care to avoid doing so. In such cases soda, bismuth, and gentian were a good old remedy. The other form was that of a chill over the epigastrium. He believed that cold air suddenly striking one through going out from a warm room would produce atony of the stomach more or less of a temporary nature. It was often experienced by people who were exposed in refrigerators to excessive cold, and who, unless they wrapped themselves up, suffered from atony of the stomach. He had seen this occur in tropical countries. For instance, a party left Cairo, where it was very hot, and travelling to Alexandria, there embarked; the sudden drop of temperature upset the digestion of most of that party. These were instances of causation of atony of the stomach which were worth investigating.

Dr. HUTCHISON, in reply, said that those who had taken part in the discussion were more in agreement with his views than he would have expected. He was interested to find that Drs. Habershon and Herschell agreed with him that, in spite of the physiologist, small doses of bicarbonate of soda were useful in stimulating secretion. The cases which Dr. Herschell had described as normal he should have classified as belonging to the over-sensibility class. The only possible explanation was that the stomach was unduly sensitive. He was convinced that that took place to a large extent in a case of hyperchlorhydria. A case which was often put down to excessive acidity was really due to over-sensibility. He agreed with Dr. Herschell's treatment of hyperchlorhydria, but such patients were often otherwise quite healthy and they very much resented being sent to bed. Although in bad cases the treatment Dr. Herschell suggested was successful, he did not suppose that in minor cases he would adopt such a treatment. He had no personal experience of the use of ergot, and did not see how it could reduce secretion, nor had he any experience of pepsinuria, but he was rather sceptical about it. He had been particularly interested in Dr. Herschell's remarks about electricity, because that was a point on which Dr. Herschell spoke with large experience. He could assure Dr. Herschell that his (Dr. Hutchison's) patients were not treated in the haphazard way he suggested, but were all handed over to a competent electrician. Latterly, in the hands of one of his colleagues, some of the atonic cases apparently improved by the application of the tri-phase sinusoidal current. The use of a diet rich in fat was perfectly sound on experimental grounds, but there were many people who had such an insuperable objection to fat that they could not be induced to take a large quantity; they would rather endure the hyperchlorhydria than swallow large quantities of fat. He was interested to hear Dr. Duncanson support his anti-ferment view, and cordially endorsed what he said about the bad effects of cold, which he believed produced gastric catarrh and gastritis.

Therapeutical and Pharmacological Section.

November 26, 1907.

Dr. T. E. Burton Brown, C.I.E., President of the Section, in the Chair.

The Therapeutics of Indigestion.

By William Murray, M.D.

I propose to lay before you some views on the treatment of a certain form of dyspepsia. My opinions are more or less commonplace, but I trust they may be of practical importance. It will be necessary to make a preliminary sketch of the kind of case I propose to treat, and make some observations of a simple nature on the physiology of digestion and the pathology of indigestion. The stomach combines many wonderful things in its various functions. More or less an osmometer, it is also to some extent a dialyser in selecting what shall and what shall not pass through its walls into the blood. On the other hand, in secreting gastric juice the stomach effects one of the most marvellous decompositions on record, inasmuch as it splits up chloride of sodium or calcium, and sets hydrochloric acid free. How it does this is, I believe, still a mystery.

We must bear in mind the stomach is a most sensitive and rebellious organ, resenting unsuitable food in unsuitable quantities, and very exacting in its demands for what is suitable and acceptable. Moreover, it is a punctual organ and expresses its wants at certain intervals with great regularity. To this end it is provided with an inward voice in the shape of appetite which is powerful enough to have been called "the monitor of man." And it not only tells us when to eat but can also tell us the quantity we need to eat and suggest the kind of food we stand most in need of—fat, to wit.

I must not neglect to refer to the psychical aspects of this matter. We all know what a powerful effect mind has on this organ, and what a still more powerful effect it has on the mind. Mind can both promote and arrest its functions, and Pawlow has shown that a psycho-physiological phenomenon occurs when one is about to eat by the preliminary secretion of psychic pepsin. There is another side to this question—what effect has the body at large on the stomach? I have written, many years ago, in the *Journal of Anatomy and Physiology*, on what I called osmotic dyspepsia, and tried to show that digestion, *ceteris paribus*, was good or bad, according to the state of the general tissues. Somatic indigestion is a state produced by eating when the blood and tissues are replete with nutriment and do not need fresh supplies. If we are tempted to eat under these circumstances nature rebels and food remains, unabsorbed and undigested, in the stomach until it ferments.

On the other hand, we may eat what we like and as much as we like without inconvenience when the tissues are starved. I remember a patient recovering from typhus who ate and digested eighteen mutton chops a day. The moral is—do not eat until the tissues are exhausted by mental or bodily labour. What is the use of our digestive tabloids, &c., when the body does not need the stuff we have thus artificially digested?

Before describing the class of case I wish to treat, let me refer to two conditions which play a large part in digestive troubles. First, I refer to the hypersensitive stomach, a condition which may have been inherited or constitutional, or induced by errors of diet and regimen, a state which resents the use of any but the most simple diet, where the patient has all his life to sail between Scylla and Charybdis to avoid catastrophes. Apropos of this, it is curious that many people with otherwise strong digestions dare not touch certain simple articles of food; some dare not touch an egg, others would half die of pork, and even milk will act like a poison to some people.

The second condition to be recognised is the "over-educated stomach," where the organ has become so refined in its tastes that it revolts at ordinary cooking. You meet with this case mostly in gourmets who have lived in great luxury, where a dinner may last for hours, while the pampered organ is filled little by little over and over again.

Let me now present a sketch to you of the class of case I wish to speak about. First, we have a weak and highly sensitive stomach, whose reserve power is small. It is therefore easily put out of order. Such a stomach has very probably been the seat of catarrh more or less frequently. These repeated attacks of catarrh sometimes result from chill, sometimes from errors of diet—not always from overeating, just as often from long fasting and irregular eating, as many overworked doctors well know from their personal experience. Sometimes the catarrh follows influenza—a bad form—sometimes from swallowing septic secretions at the end of a bad cold, or from suppurating teeth and gums. Such a stomach will often have been the seat of fermentation and flatulent distension, so often that its lax cardiac end becomes dilated more or less permanently. All this may happen without much severe pain, but it is almost always accompanied by severe mental depression, which in its turn depresses the stomach and awakens the neighbouring nerves to a splanchnic sensation of much misery. I need not dwell on the rôle of symptoms in such cases, they are only too familiar to all of us. We must now consider their treatment.

First of all, how shall we diet a patient in this condition? We must elicit what has hitherto agreed or disagreed with him, also when and how often he eats, *e.g.*, whether he is frequently nibbling to stave off the craving for a too late meal, whether he smokes too much and on an empty stomach, and what his alcoholic tendencies may have been. Above all we must find out whether he chews his food properly or bolts it like a hound. His teeth, of course, must be examined, especially his molar teeth, and we must see whether they meet when closed or miss each other in masticating—especially in the case of boys and girls and old people. Note also how he clothes his stomach, whether his trousers and waistcoat are cut too low to cover the epigastrium, an important point for a motorist, who is especially liable to gastric chill. We shall often be able to eliminate a dominant factor by these enquiries. We must also find out whether the various components of his meals are complemental and likely to agree together, and whether our patient goes to bed with too full or too empty a stomach; both are bad for these people.

To begin the day. Shall we allow an early cup of tea, well diluted with milk? I think we ought to do this, for a weak and empty stomach is always uneasy, and such patients often awake early and pass a miserable hour. Moreover, an empty stomach is liable to fill with flatulence. We must therefore help this weak stomach by a warm nutrient drink. Warm water is too harsh, milk alone is too heavy, and patients tire of Benger, but they seldom tire of the early cup of weak milky tea.

We have now got him down to breakfast. What shall we give? Let him have two table-spoonfuls of porridge, with plenty of milk, as a first step. The way is now paved for frizzled bacon, with the superfluous fat frizzled out of it by superheating. Taken with thin-cut dry toast there is nothing so good as frizzled bacon. Next comes the lightly-boiled egg, either insinuated among the bacon or alone, or the bacon can be eaten with white fish and a pinch of cayenne pepper. Finish with marmalade and toast, and your patient has got a good, wholesome meal. I trust you will pardon my attention to these simple details; I regard them as all-important.

Let us now pass on to the lunch or mid-day dinner. Mutton, well kept and well roasted, is our best "stand-by" for lunch, but bear in mind the mutton *must not be fresh;* it must be kept as long as it *will* keep, or it will not digest. In frosty weather with a good larder this is easy enough, but in hot, muggy weather, as most larders are very faulty, it is difficult. For old people, or people with bad teeth, the mutton should be finely minced, and it is safer to give with it well-boiled rice instead of fresh green vegetables or potatoes. At lunch this weak stomach is better without the burden of a pudding, and a crisp biscuit with a morsel of good cheese ought to complete the meal. Liqueur brandy, faintly diluted, is here of great value as a cordial beverage.

So far we have got on very well, but afternoon tea is our *bête noire.* People will not take it properly; if they dine early they eat too much bread and sweet cakes with it, *i.e.*, before the stomach is empty, and if they dine late, they take it too soon before dinner. It should be taken at least three hours before a late dinner. One cup with plenty of milk is enough. *Homogenised*[1] milk, where you have the cream inseparably diffused through the milk, is far better than cream or milk separately—no scum on the top then.

We must now consider the late dinner on which the patient is to subsist until the next morning, and we must therefore try to get in a good meal by hook or crook. As there is probably a lack of appetite, we must tempt the stomach; a wholesome flavour tempts it by stimulating the collateral senses. We therefore place some lightly-seasoned and well-flavoured soup before our guest, and limit him to a teacupful, with thin toast, dry and crisp, to break up and mumble as he dines, after which half a glass of dry sherry or a nip of fine old liqueur brandy; this gives a fillip to the weak organ and helps it "over the stile" to give a replenishing stream to the exhausted body. Now comes the course of white fish and a sprinkling of cayenne, one of our best gastric stimulants, and harmless. The entrée of sweetbread and minced chicken is good if your chicken has not been fed on garbage, and if your sweetbread is from the right kind of animal and the right part of it. The meat course should again be of the tenderest: well-kept mutton, or a steak which has been suspended by an antiseptic thread to isolate it, in a dry, cool place, for *three* weeks; then it melts in the mouth and appetises as it digests, so that our patient is in danger of eating too much. A light rice pudding is quite suitable, it does not cause flatulence. Our patient should finish his dinner with a hot biscuit, buttered and peppered, and a nip of liqueur brandy. This final nip keeps all in order.

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[1] It is made by driving the milk against an agate slab by a 16 h.p. engine at the Cumberland Dairy.

Having tried your patience with these homely details, we now come to the question, How are we to treat these stomach troubles by drugs? There is no department of health which is so dependent on the proper administration of drugs as that which pertains to digestion. The easy access of our remedies to the seat of trouble, their direct and almost immediate action, and the rapid determination of their effects combine to make these organs a sphere of observation of intense interest.

Let us now suppose we have to treat this hypersensitive, catarrhal, dilated, irritable and feeble stomach by medicines; where shall we begin to supplement the plan of diet I have laid down? Suppose we begin *primo mane* after the patient has had his weak milky tea or Benger. I give him a teaspoonful of vegetable charcoal, best taken just after the patient has brushed his teeth. In all probability the process of tooth-brushing has developed slight retching, with consequent churning action of the stomach, for the fauces and pharynx are very sensitive in these cases. The charcoal purifies the mouth and soothes the pharynx and gullet, and afterwards does the same thing to the stomach. He now feels more comfortable, having probably eructated a volume of gas which has accumulated in the empty stomach. His breakfast proper must be eaten in a warm room, or in a top-coat if the room is not warm. Ten minutes after breakfast, when the organ is labouring to digest its first meal, we must come to its aid by giving a "digestive ferment." Do not rely on ordinary pepsin *uncombined;* it often smarts a tender mucous membrane; qualify it by the addition of that most valuable of all gastric sedatives, "the oxalate of cerium." Cerium neither retards digestion nor impairs appetite, nor does it constipate like bismuth. Thus we give 10 grains of pepsin and 8 grains of cerium soon after breakfast. If there is much tendency to acidity I do not give ordinary pepsin, but fall back on lacto-peptin, which suits these sour stomachs much better, and is a very good digestive. If there is morning retching I advise a 10 grain dose of cerium with pepsin or lacto-peptin *before* breakfast instead of *after* the breakfast.

You will find the above treatment very suitable in the irritable dyspepsia of phthisis, and it gives the patient a chance of assimilating cod-liver oil and the malt extracts, and agrees well with hypophosphites of lime and soda, &c., all of which are best essayed during the early forenoon.

We now proceed to the ante-prandial treatment or the dose before lunch and dinner. We will find nothing better for this than the pepsin and cerium powder, but it should be given *before* and not *after* the meal. I have often tried it after these meals, and never get the same results as the ante-cibum dose gives me. It seems to whet appetite and stir up the secreting action of the stomach.

Should there be laxity of the bowels, with mucous catarrh therefrom, we can give the charcoal again before dinner with the oxalated pepsin. (Please do not give any of these ante-prandial remedies in the tabloid form; powders, as it were, dust the gastric membrane and act at once, and the digestive tabloid does not always dissolve quickly enough.) Should the patient suffer from nausea and sickness, he must have recourse to another digestive ferment of great value, *viz.*, "ingluvin." It may be given alone with good effect, but it, like all other digestive ferments, acts better in combination with the oxalate of cerium. In such cases this combination often acts like a charm, and thus saves the situation.

Now let us go back a little and provide for a failure of these remedies. If the patient is still uneasy after meals, and if there is pain and weight after eating, with flatulent distension, it is evident our powders do not meet the case; we will therefore try another more decided digestive sedative. I then suggest a mixture of pepsin, hydrochloric acid and tincture of Indian hemp, a combination from which I often get good results, especially if bismuth be added in bad cases. It may be given before food, but it is better to give it after meals in most cases. When this mixture has soothed the stomach and given it time to recover its enfeebled powers, we may substitute 3 drops of Tinct. Nuc. Vom. for the Indian hemp, and thus get a good form of tonic digestive, which helps these cases immensely. This tonic form of digestive should be given after *breakfast* and *before dinner* and lunch.

Suppose we are dealing with one who takes a midday dinner, and supposing that digestion of a fairly good meal has been achieved, we may allow tea with dry toast as a kind of test meal to show us how far the stomach can be trusted. If it distends with flatulence, and if sour eructations follow the tea, we know that imperfect digestion has occurred, and we must remedy these discomforts by a dose of hyposulphite of soda, carbonate of soda, rhubarb, and ginger, which corrects acidity, stops fermentation, and dispels flatulence at one fell stroke.

We have now run the gamut of our ordinary routine treatment of these cases. It is a long rôle, but very harmless, and it is wonderful how patients will take to it and stick to it. I always tell them they must devote themselves to this treatment as a serious business, and they mostly get their reward if they do so. There are, however, many exceptions to these rules of treatment. For instance, there may be indications of ulceration in a major or minor degree, in which case we must, of course, give a dose of bismuth and morphia ($\frac{1}{20}$ gr. is enough at first) combined with soda and magnesia, and a drop or two of chloroform—never give spirit of chloroform, it nauseates. Then again, when undue flatulence prevails, we must remember the value of assafœtida carminatived with these essential oils, and when the secretions are out of order (to use an old-fashioned expression) we give a mild corrective warm pill of pil. hydrarg., rhubarb, capsicum and opium, and oil of mint. This puts the secretions right without trouble to the patient, and is far better than raking out the bowels with colocynth and such-like. We must beware of tonics in such cases, they mostly do harm, especially if given more than once a day, namely at 10 a.m.

Time fails me to dwell further on this subject; it is almost endless in its details, and one has to follow the vagaries and idiosyncrasies of the stomach in many devious paths. I should, however, like to add one remark in favour of the *black oxide of manganese*, a much-neglected remedy. It soothes the stomach like bismuth and cerium, and seems to build up the mucous membranes, and at the same time it acts like a ferruginous tonic in anæmia. It is also an excellent emmenagogue, so that if we have to treat an anæmic young woman with an irritable stomach which won't tolerate iron, we have here a remedy which meets all these conditions very effectually.

And now I must draw to a close, with many apologies for adding but little that is new on this subject. I have only attempted to confirm what is already pretty well known, by giving the results of a personal experience of nearly fifty years duration, so that my views are probably old-fashioned. We have each to work out an experience for ourselves, probably on better lines than mine, for the evolution of our treatment must follow the evolution of our better knowledge of all that concerns the welfare of man and the ills his flesh is heir to.

DISCUSSION.

The CHAIRMAN said they had listened to a most practical and interesting paper, and he felt sure that many of them could give some information on the subject if they would discuss it.

Dr. SOPER said there was only one thing in which he would venture to differ from the reader of the paper, and that was, he himself was under the impression that of all the things that a human being put into his stomach, the most absolutely indigestible was frizzled bacon, because as soon as bacon was heated to a high temperature of over 212° F., it released what it did not possess before, viz., a fatty acid, which was most offensive to the stomach. He would also like to ask Dr. Murray how he would administer chloroform, if not in the form of spiritus chloroformi, as it did not easily mix. Another point in which he agreed with the lecturer was, he presumed that when he gave patients black oxide of manganese, it was with the idea of trusting to the presence of chloride of sodium, and so getting free chlorine, which he (the speaker) felt very satisfied was most beneficial in all cases of indigestion. As an old practitioner of about fifty years standing, he thought he might say that the rising generation did not know the advantages and virtues of *strong* nitro-hydrochloric acid. He advised them never to give the dilute acid, but always the strong nitro-hydrochloric acid. He knew it was a little troublesome to keep in the surgery, because it had a habit of driving the stopper out of the bottle ; but it contained a certain amount of nascent chlorine, which could thus be administered to the patient, that one did not get with the dilute acid.

Dr. MURRAY said he would like to say that the great point was to thoroughly roast the fibrous tissues in the bacon. It was that horribly stringy stuff, which was not properly cooked, that caused the most trouble, but when it was superheated it became like the "crackling" outside pork, a digestible article instead of an indigestible one. If they examined a vomit from a patient of weak digestion, who had been eating bacon, they would find the strings of bacon absolutely untouched by the gastric juice. Personally, he did not understand the chemistry of these fatty acids, but he believed it was a subject very well worthy of attention. It was for the sake of getting rid of the indigestible article in the bacon that he recommended the frizzling and superheating of it.

Dr. CECIL WALL said he would like to ask if it was really true that the fatty acid set free in the cooking of bacon was deleterious, because of recent years—he only spoke from the laboratory point of view—he believed people who had investigated the use of cod-liver oil had shown that cod-liver oil was more digestible if free acid was present. And working on these lines, when cod-liver oil became dear, a substitute, consisting of pure oil and free fatty acid, was put on the market ; at the hospital where he practised they had used a substitute, which contained free oleic acid up to 5 per cent., and it was easily digested. Therefore he wondered whether it was quite true that the presence of free fatty acid was the reason for the indigestibility of bacon.

Dr. J. GRAY DUNCANSON said he was quite sure they all felt deeply grateful to Dr. Murray for the very interesting paper he had brought before them, and more especially since Dr. Murray had journeyed from Cumberland in order that he might meet the Section, in which he took the greatest interest. He entirely agreed with Dr. Murray with regard to the value of chloroform in such cases as he had described, not the spirit of chloroform. He simply put a few drops in the bottle, and he found it sank to the bottom and did not rise to the top as had been suggested by one speaker ; and if the patient was instructed to shake the bottle it would mix very satisfactorily with the medicine. He concluded by moving a hearty vote of thanks to Dr. Murray for his interesting paper.

Dr. INCE seconded the proposition, and said he thought it was very appropriate that they should have one of the old ones and one of the young ones to propose and second the vote of thanks. It was a great pleasure to see a gentleman like Dr. Murray taking the interest which he did, and coming all the way from the North of England to visit their infantile Section of Therapeutics. It was well, perhaps, that he had had an opportunity of attending, so that he might leave his name on the records of the Society as having delivered one of the most interesting papers they had heard since the Society was established. He was quite sure they represented the wishes of all present when they proposed that a hearty vote of thanks should be given to Dr. William Murray for his kindness.

Dr. HERBERT FRENCH said that if the discussion was not closed, he would like to ask Dr. Murray three questions. First, he asked for his own information—in his ignorance he had not heard of the word ingluvin before—he would like to have particulars of what it consisted of and what was the dose. The second point was with regard to the constipating effect of bismuth. At Guy's Hospital Dr. Hertz had lately been experimenting with bismuth in connection with X-ray examination of the alimentary canal. He had been giving bismuth oxycarbonate through the mouth, and he had given up to two, three, and four ounces at one time, and, in spite of doses of that size, he had been unable to verify its reputed constipating effect. The third question he wished to ask was in connection with smoking. Dr. Murray had not mentioned it in his treatment, and he (the speaker) would be glad to know what his views were; did he disallow it, or did he allow it, and if so to what extent, in this particular variety of dyspeptic case?

Dr. MURRAY, in replying, said he was very glad Dr. Duncanson had corrected the slip of the tongue about chloroform floating and not going to the bottom of the bottle. The question of the use of nitro-hydrochloric acid was a most important one, and he felt quite sure that the old-fashioned remedy of using the fuming acids had fallen into abeyance. He remembered once calling in Dr. Begbie, of Edinburgh, in the case of a man whose functions were out of order and whose body was almost broken up—he had ulcerated legs and many bad symptoms—and the doctor said, "Give him strong nitro-hydrochloric acid." He therefore put him on it, and in a month the patient was on his legs again. In reply to Dr. French's question about ingluvin, he believed it was made in America from the gizzards of birds. It was a wonderful digestive and was given in the form of a powder in 6-gr. doses. It went well with cerium and acted very nicely on the mucous membrane and allayed sickness in a very marvellous way. With regard to the question of smoking, all he could say was that, being a smoker himself, he could say that it was a bad thing on an empty stomach, and it was a bad thing for anyone who was going to make a speech to smoke beforehand. Referring to pepsine, he had been trying experiments for some years, on and off, as to the pepsine of fish. If some pharmacist would discover what digested the awful stuff that went through fish he was quite sure it would be a valuable discovery. He remembered being at a hatchery and seeing a 4 lb. trout which had swallowed the back sinew of a horse, part of which was sticking out of the fish's mouth, part was in the fish, and part of it protruded from the anus in filament. It was as thick as his wrist, and he said to the man who had charge of the fish, "Why don't you kill that thing?" He said, "When we are feeding them on horse-flesh they often get hold of the back sinew, and it takes about three weeks to digest." He believed if they could only discover what that pepsine was, it would be a very good thing. Messrs. Brady and Martin had told him that so far as they could make out it was alkaline pepsine. At any rate, it seemed to him it must be a very remarkable pepsine to digest the back sinew of a horse. In conclusion he was very grateful to them for the kind way they had received his paper. He had almost retired from practice, and he found it most refreshing to go back to his work and bring some of his results before younger and fresher minds.

Some Tropical Diseases and the Remedies required for their Treatment and Prophylaxis.

Notes on a Demonstration.

By James Cantlie, F.R.C.S.

The subject I bring before you is one that deals with our ever-advancing knowledge of pathology, and especially with the branch of medicine which, for want of a better name, we term "Tropical." For the past twenty-five years bacteria have well-nigh usurped the attention of investigators, but at the present time and for some time to come the protozoa and the diseases they cause in men and animals demand, and will continue to demand, a foremost place in pathological research. I am to show you a few lantern slides, not with the idea of discoursing upon Tropical diseases and parasites which you are no doubt already familiar with by the publications in medical journals, but I show them really as a help to understand the difficulties which arise in dealing with the destruction of the protozoal parasites which constitute so large an element in the etiology of disease in warm climates.

The first group of parasites I bring to your notice is that of malaria. The parasite occupies the red blood corpuscle, it is safely ensconced there, and were it content to remain as an intracorpuscular entity the drug that has proved fatal to its existence—quinine—would, so far as we know, be inert. It is only when, for purposes of the continuance of its species, the parasite bursts from its corpuscular environment and its spherules lie naked in the blood-stream, that quinine is effective. The escape of the parasite from its corpuscle is synchronous with the febrile attack characterised by cold, hot, and sweating phases, and it is the knowledge that the febrile development and the escape of the parasite into the blood plasma are related that determines when the drug may be given with greatest benefit. When quinine is given as a prophylactic, a question arises how often should the dose be administered. To give it daily is by some regarded as detrimental to the blood itself, and by others the persistent administration is held to produce a tolerance of the drug which weakens its power as a protozoacide. A scientific basis for administration is vouchsafed to us, however, by the fact that quinine in a full dose continues to circulate in the blood for three days after being taken, and therefore, if, say, 15 gr. of quinine are taken every fourth day, we have a prophylactic measure at once safe and rational. Quinine, however, is limited in its power of attacking the malaria parasite. In early infections only are its benefits markedly apparent, but when chronic or latent malaria supervenes its potency is doubtful. The reason for this is evident from the fact that the cycle of life of the parasite changes. The asexual reproduction which proceeds within the human being would not serve for the continuance of the species of the parasite indefinitely; this is provided for by a true sexual cycle which necessitates an extra-corporeal life in an alternative host which we know to be the mosquito. The preparation for this sexual cycle is attended by alterations in the parasite itself and in the corpuscle which envelopes it. In this phase of the parasite's life quinine has but feeble powers, and its administration is attended by disappointment. The question then comes, can quinine be supplemented by any other drug or drugs at our command? It was the custom of our immediate predecessors in the practice of medicine to affect lengthy prescriptions in the treatment of almost all diseases; it is the prevailing custom of to-day, however, to withhold drugs in the treatment of disease, or, when given, to prescribe one drug by itself. Of course, the practitioner of to-day thinks he is right and condemns the aggregation of drugs which characterised the pharmacy of our predecessors.

That we are right and that our predecessors were fools is no argument, and in fact will not stand the light being thrown upon the treatment of disease by the pathology of the past year or two. In England the old ague powders were composed of quinine, arsenic, and opium, and the exhibition of the powders was invariably preceded by a dose of calomel.

It is extraordinary how frequently calomel is "required" as a preliminary to the use of specific or empiric medicines. Is the use confined to unloading the liver, cleaning the tongue, &c., or is the good it does more of a parasiticide order? I believe the latter, and in the treatment of malaria we find quinine, arsenic, opium and mercury combined in the remedy for chronic malaria. Malaria has disappeared from England, and we say, perhaps quite correctly, that the disappearance of malaria in England was due to the country being efficiently drained and the swamps where mosquitoes bred done away with. We ascribe nothing to the use of drugs, yet in the Tropics quinine is advocated as one means of preventing and exterminating malaria.

Quinine in this respect is, however, a failure for several reasons: one reason may be because it is improperly given; that is, it is administered as a rule by itself. In England malaria was got rid of whilst it was the custom to combine quinine, arsenic, opium, and mercury, and although we cannot say it was because of this combination, yet did it disappear coincidently with the exhibition of this combination. This is no doubt a defective argument, but I would urge the advisability of a return to the custom of prescribing quinine with the adjuncts found by experience to be useful. I would cite the compound known as Warburg's tincture as an example of the benefits accruing to polypharmacy. As to the potency of this mixture I am convinced, as to its superiority to plain quinine in malaria of long standing I am positive, and I believe its efficacy is due to the accessories to quinine which it contains. The learned President of this Society informs me that Warburg's tincture, without the quinine it contains, was used, under another name of course, in the time of Mithridates as a remedy for fever, and that Warburg of Vienna merely added quinine to the compound and baptized it "Warburg's tincture." In the matter, therefore, of even so marked a specific as quinine for malaria, I believe it is by itself less potent than when combined with some such accessories as one finds in Warburg's tincture, or in the old ague powders we used in England.

The next parasite I would draw attention to is the filaria, a blood worm whose presence is the cause of many conditions grouped together under the name filariasis. The parent worms become ensconced in the connective or lymphatic tissues of the body, and their progeny are to be found in the blood as small active worms. This parasite also requires an extra-corporeal existence in a mosquito (Culex) for the continuance of its kind, and it was the study and recognition of this phenomenon by Sir Patrick Manson long before the mosquito-malaria theory was announced, and even before the malaria parasite was discovered, that formed the basis of thought by which Sir Patrick conceived the mosquito-malaria theory. The study of this parasite in relation to its life-history and to the mosquito is one fraught with the deepest scientific interest. The variety of filaria known as the *Filaria nocturna* appears in the peripheral blood of man only at night; it comes to the surface at the time the Culex mosquito in which it is to pass its extracorporeal cycle is about, namely, at night. It comes to the surface no doubt in order that it may reach the mosquito's stomach when the insect bites man and draws blood. Can we assume that an embryonic worm, with its tissues differentiated but to a small degree above the level of a mass of protoplasm, has the intelligence to know that by coming to the skin it can reach the mosquito's stomach, or is it the mosquito that has the super-intelligence to bite man at a time when the worm is in the peripheral blood? The worm, however, is not necessary for the existence of the mosquito, but vice versa. We must therefore assume that the intelligence is with the worm, however it may astonish us and however it is to be explained. Such intelligence, however, is seen throughout Nature. The flowers which attract moths throw out their odours at night so that the moths settle on them and carry their pollen to impregnate other flowers; the flowers which butterflies, bees, &c., affect, expand during the day, so that these "day" insects may perform a like function. The owl is awake when the mouse is about, the fox when his prey is asleep; the parasite in latent malaria seems buried in the tissues during winter months, but appears in spring in the peripheral blood when the mosquito becomes active. To ascribe these phenomena to chance is of course absurd. An extraordinary example of Nature's provision for the continuance of a species is shown in the case of a guinea-worm. The embryos I show on the screen gain access to the stomach of man, hence they proceed on attaining maturity usually to the lower extremities, where the female reaches the surface by causing a minute aperture in the skin. On water being applied to this aperture the parasite pours forth a milky fluid, which on examination is seen to contain many embryos. Why should the parasite affect the lower limbs as a rule? Because it is necessary that its embryo should reach water, and as in countries where the guinea-worm prevails the natives wade in water with bare feet, the female parent lies in wait until she finds a watery surrounding, when she sheds her young. But the female guinea-worm may reach any part of the human body, yet will it be found that it is always a part where she has a prospect of depositing her young in water. Thus in the case of the washerman the worm may affect the upper extremities, or in the case of the water-carrier it may be in the back or shoulders, for the water-carrier in warm climates usually places the skin in which he carries water on his back or shoulder. These phenomena constitute one of the most interesting Nature-lessons it is possible to conceive.

The curative drug treatment of filaria and of guinea-worm has not yet been discovered; we do not know how to destroy the parent worm by internal remedies. That such treatment is not possible I do not believe. We know that quinine destroys the malaria parasite, that the spirochæte of syphilis yields to mercury, and Plimmer has shown that antimony in animals destroys the trypanosome. The trypanosome parasite is at the present moment occupying close attention, owing to the fact that it is the cause of sleeping sickness. I show you several trypanosomes on the screen, but I cannot delay to analyse or differentiate them. I only wish to draw attention to the treatment of trypanosomiasis—in other words, to the destruction of the parasite. Atoxyl, itself a compound of drugs, has gained considerable reputation in sleeping sickness. By its use the trypanosomes disappear from the blood, but only for a time; many of the aniline dyes have a similar action, showing that in these drugs a certain parasiticide action obtains, but that an evident tolerance of the drug declares itself, and the parasite reappears in consequence. Many metallic substances have been tried, including mercury, arsenic, copper, &c. Arsenic seemed the most potent, and in the form of atoxyl has had partial success. Mercury, however, has been found a necessary adjunct, and some declare that by atoxyl the trypanosomes are driven forth from the blood, and then, by the subsequent exhibition of mercury, the parasites are destroyed. This implies a polypharmacy of definite import, and causes one to review all one's treatment by drugs, and to favour the compound mixtures of our predecessors. But Mr. Plimmer has recently found that in antimony we have a drug which is effective in destroying trypanosomes, at all events when tried experimentally in animals. Antimony has been a neglected drug for many a day; its success in times gone by has proved its undoing, for its efficacy in one or two diseases led to its adoption for many ailments, and it thereby fell into disrepute. Now it would seem that antimony is again to assert itself, and we may, we hope, soon hear that the drug is to be successful in killing the trypanosomes of man.

A study of the protozoa, and of the relation of disease in domestic and other animals to those of man, is leading us into a peculiar channel of thought. The mosquito is to be exterminated to free us from malaria; the rat is to be similarly dealt with, because by its fleas it infects man with plague; the crocodile, according to Koch, ought to be destroyed, as it is the extra-corporeal host of the trypanosome of sleeping sickness, and so forth. Because the dog gave man hydrophobia we did not find it necessary to destroy the canine species, but only to muzzle him for a time, whereby not only has hydrophobia disappeared from man in these islands, but also rabies from the dog. It has not been found necessary to destroy the horse to rid us of glanders, nor the pig to free us of trichina. So it may be possible, by other methods than by the extermination of whole species of animals, to find means of holding disease in check more in keeping with Nature's plan of created beings.

DISCUSSION.

Professor CUSHNY said he would like to congratulate Dr. Cantlie on the exceedingly interesting paper he had read, and also to congratulate the medical profession generally on the fact that in tropical diseases animal parasites were found to play so large a part, for they seemed to be much more susceptible to the action of drugs than the bacteria. He thought it might perhaps interest the members of the Society if he told them how they came to use antimony. A sub-committee of the Royal Society had been working on the therapeutics of trypanosomiasis for about a year, Mr. Plimmer and Dr. Thompson carrying out the experiments, while his rôle had been to advise on the pharmacological aspects. They had tried a number of lines such as quinoline and dichlorbenzidine bodies without success, and later numerous arsenic compounds such as atoxyl, which undoubtedly had a beneficial action. Thinking over the matter from the pharmacological side, they thought they would try antimony as the nearest element to arsenic, except phosphorus, which, of course, was impossible. Their difficulty was in what form it was to be given, as it was so irritant that it was used as a pustulant and emetic in former times, and it did not therefore seem possible to use the ordinary preparation. He therefore made a compound of antimony and glycine in the hope that it would be less irritant. At the first injection of this body the parasites became reduced in number, and on making a stronger solution it was found they disappeared altogether in the course of about two hours. About that time he was called away from London, the supply ran out, and Mr. Plimmer and Dr. Thompson had to get some other compound of antimony. They tried tartar emetic, and they found that just as good, or better. At first they found the rats died, and so they used the sodium salt. As a matter of fact, it was now believed they died from the excessive destruction of the parasites. The method now was to give three or four small doses of tartar emetic, or its sodium analogue, in order to lessen the excessive destruction of the parasites, and the results had been extremely satisfactory. From the arsenic compounds they found not more than 5 per cent. of the rats really survived, as the parasites recurred in the others in the course of a fortnight. After a fresh dose they again disappeared, to come back in a week or so. About 95 per cent. died when atoxyl was used; but since antimony had been used he believed that out of the last twenty-five rats that had been treated twenty-three had no recurrence, so that the prospects were exceedingly good, and he believed the treatment might prove of value in the case of sleeping sickness. He did not know whether Dr. Cantlie could be persuaded to give them any information with regard to men under treatment. If antimony proved to be of value, as they hoped it would, it was on the whole rather an achievement for purely experimental work.

Mr. MABEN proposed a vote of thanks to Mr. Cantlie for his paper. Though he could not discuss the subject of the paper, he wished to refer to the possibility of immunizing following the repeated administration of quinine. An instance had come within his own knowledge where a lady, somewhat advanced in pregnancy, had for two months suffered from very severe headaches, which nothing would relieve but hypodermic injections of morphia. The result was to render the boy who was subsequently born practically immune to the effects of morphia. The boy was also born with nystagmus, which was still very evident when he was labouring under excitement, his eyes oscillating with great rapidity.

Dr. J. GRAY DUNCANSON seconded the proposition, and said it was very important to hear questions concerning malaria treated by a man who knew something about them. Nowadays people travelled abroad more than they used to, and one saw a great many cases in this country. It was therefore important that they should know how to treat such patients as might come under their notice. Quinine in certain chronic cases seemed to lose its effect after a time, and then a combination of quinine and arsenic appeared to be most beneficial. He begged to ask Dr. Cantlie if he had ever found quinine to cause hæmoglobinuria as stated by some writers. The speaker could not believe that such took place, and instanced a very severe case of hæmoglobinuria which occurred in a man who had not taken 60 gr. of quinine in the whole of his lifetime.

Mr. CANTLIE, in replying, said he was very much obliged to them for the way in which they had listened to his crude remarks, which had been put together very hastily. He had not said all he wanted to, and one of the things he wanted to say was that a change of drugs, as well as a change of foods, was most important in connection with the treatment of disease. He found that if patients suffering from any form of intestinal flux, especially in such a disease as sprue, were fed solely upon milk, after a time the milk lost its beneficial effects, and if a diet of meat only were tried, it also lost its effect. But if they gave meat one day and milk for, say, two or three days, the result was very beneficial. He had several people under treatment who had a milk diet one day in the week and the other days they kept to meat, with marked benefit. The only explanation of this seemed to be that suggested to Mr. Cantlie by Sir Lauder Brunton, namely, that the pathological germs in the intestine could in time become accustomed to and succeed in growing in any medium. When milk alone was given, the germs at first partially succumbed, but after a time they became accustomed to their environment and recovered and multiplied. The same took place when a change was made to a pure meat diet. By the alternate use of changed diets the disease germs would in time become extinct.

Therapeutical and Pharmacological Section.

December 17, 1907.

Dr. T. E. Burton Brown, C.I.E., President of the Section, in the Chair.

The Action of Digitalis on the Human Heart.

(ABSTRACT.)

By James Mackenzie, M.D.

The communication consisted of a brief résumé of the result of an enquiry into the action of digitalis on the human heart. It was pointed out that there was no principle guiding the prescription of digitalis, each man being influenced by imperfect deductions drawn from experimental work joined to his own individual experiences, the result being that digitalis is often prescribed in cases where it can do no good, but happily it does little harm, as it is far from having the effect usually assigned to it. The question of the relative efficiency of the various forms in which digitalis is prescribed was enquired into and it was found that all forms of the drug were equally effective in certain cases and equally ineffective in others. Further enquiry showed that the supposed uncertain action of the various preparations was really due to the varied lesions of the heart, certain hearts being extremely sensitive to the action of digitalis, while others were unaffected by it.

In cases where the rhythm of the heart was normal, digitalis had little effect upon the rate. Even in cases where it was pushed till sickness, vomiting and diarrhœa ensued, the heart's rate was only slightly affected, and the arterial pressure was not raised. When there was a quickening of the heart from tuberculosis, rheumatic fever, or pneumonia, no effect on the heart's rate resulted from pushing digitalis till sickness resulted.

The cases which benefited from digitalis were those in which there was dilatation of the heart. Indeed, so manifest is the relationship between the beneficial effect of digitalis and dilatation of the heart that it may be laid down as a guiding principle that digitalis is of no avail unless there is dilatation of the heart. A large number of cases of heart failure with no dilatation were taken and digitalis pushed until sickness was produced, but no improvement was felt by the patient. The fundamental reason for this is that dilatation only occurs when there is a failure of the function of tonicity—dilatation being, in fact, the outward and visible sign of depressed tonicity—and digitalis has a remarkable effect on the function of tonicity, and in restoring this function it places the heart in a better position for carrying on its work.

When there is a delay in the passage of the stimulus for contraction from auricle to ventricle, digitalis has a very powerful effect upon the heart. This delay is measured by tracings from the jugular pulse, when the time is measured from the beginning of the wave due to the auricle to the beginning of the carotid pulse. Normally, this period is one-fifth of a second in duration. As a rule, an increase of this period means an interference with the conductivity of the fibre joining auricle and ventricle. Digitalis may increase the length of this interval to such an extent that the stimulus for contraction fails to reach the ventricle, with the result that while the auricle beats regularly the ventricular systoles drop out at intervals, sometimes so frequent that only every second auricular systole is followed by a ventricular systole. This was shown to be due to excessive stimulation of the vagus, and a great many different forms of arhythmia may arise from the effect of digitalis upon the vagus in certain forms of heart disease.

It is well known that some hearts are very susceptible to digitalis, slowing of the rate taking place with small doses. An enquiry showed these cases to be of a particular kind, namely, where the contraction of chambers of the heart no longer followed the normal sequence, but where the ventricle started the rhythm, the auricle following the ventricular lead and contracting during the ventricular systole, "the ventricular rhythm." This ventricular rhythm is recognised by that continuous irregularity of the heart's action which is so common in the later stages of rheumatic heart and arterio-sclerosis. It is on the former class of cases that digitalis has such a wonderful effect. One can often play upon such hearts at will, quickening or slowing the rate by exhibiting or withholding the digitalis. On the other hand, in the irregular heart consequent on arterio-sclerosis, digitalis has little effect on the rate, even when pushed so as to cause sickness. Why there should be this susceptibility in the one case and not in the other it is difficult to understand, but the difference is so marked that the fact deserves consideration.

DISCUSSION.

The PRESIDENT (Dr. T. E. Burton Brown) thanked Dr. Mackenzie, in the name of the Section, for his very interesting paper. Digitalis was said to be the first medicine used rationally, but he thought it had just been shown that it required a little more reason still among its users.

Professor CUSHNY said he had been very much interested in Dr. Mackenzie's work, and appreciated the many suggestions which he had derived from it. The action of digitalis had been worked out largely on animals, and there was difficulty in making further advance from the pharmacological side, and it would now be well to get some accurate measurements from the bedside. A few years ago he was able to say that what was needed in the therapeutics of digitalis, and indeed of many other things, was not so much animal experimentation as clinical observation. That was now being rapidly supplied by Dr. Mackenzie and the school which he had originated, by the accurate observation of what went on under the influence of digitalis. The paper was an admirable example of how much new information could be obtained in regard to the action of drugs by ordinary clinical methods. That was needed in regard to almost every drug. It was very unfortunate that too many practitioners were accustomed to make their diagnosis and prescribe some drug and then evinced no further interest in its effects. Much interest resulted from watching the action of a drug, and there was still much valuable therapeutic information derivable from it, as had been proved by the paper they had just heard. The action of digitalis was an extremely complicated one. Even when the hearts of animals were exposed and the same dose of digitalis was given in the same way in two successive experiments, it was seldom that quite the same effect was seen. The reason of that was, that digitalis acted upon two practically opposing functions of the heart. It acted first upon the heart directly and increased its activity in almost every direction. On the other hand, it acted as a bridle on the heart: it not only used the spur, but it also tightened the reins by stimulating the inhibitory function. What any heart was going to do under those two influences it was very difficult to tell. For example, in one heart the conductivity might be increased, *i.e.*, the inhibition was very inactive; but in another case inhibition prevailed over the direct action, and the conductivity was lessened. When one knew the curious effects to be obtained from digitalis in animals, one could only admire the daring of the physician in trying it on a diseased heart as a routine treatment. He did not deny that the drug was of immense value in diseases of the heart, and it was one of those drugs whose absence would be seriously felt in therapeutics, but there was yet much to learn as to how it acted in cases of heart disease, and in what conditions of that organ its influence was beneficial.

Dr. H. C. CAMERON said he had been taught, and had been accustomed to read, that some time must necessarily elapse before the action of digitalis became apparent. This was said to be the justification for the complicated prescriptions which were sometimes given in cardiac disease. It was said that evidence of the action of digitalis must not be expected for forty-eight hours or so, and therefore other drugs must be given as well which should act immediately. He would like to hear from Dr. Mackenzie how soon after beginning to give digitalis he was able to see those changes which he described. From what Dr. Mackenzie said, he imagined the changes were observed much sooner than was generally taught.

Dr. MACKENZIE, in reply, said he had tried to find out how soon digitalis would affect the heart. He found that it depended upon the nature of the disease, some hearts reacting within forty-eight hours, and others showing no change for two or three weeks.

The Reminiscences of an Apprentice Fifty Years Ago.

(ABSTRACT.)

By WILLIAM SOPER.

DR. SOPER said that he had been reading lately the life of Sir William Gull, and in it he had found the following sentence: "He thus escaped, most fortunately for him, the many disadvantages attaching to the apprenticeship system then in vogue—a relic of the dark ages, happily now extinct, whereby men obtained a practical knowledge of bottle-washing and dispensing, and learned sufficient rule-of-thumb midwifery, minor surgery and drugging to enable them to act as qualified assistants, relieving their principals of much drudgery, and paying for the privilege into the bargain." His own experience was very different. He thought of the old days of apprenticeship with joy, and felt absolutely sure that time was not lost, but a vast amount of knowledge gained to prepare for the battle of hospital life. In a paper full of interesting personal experience, he described the life of an apprentice of fifty years ago. The practice of blood-letting was still much in vogue, and he had seen persons with the scars of at least a hundred incisions. In many cases he was sure that it was of great benefit to the patient, and he was still more sure that blood-letting would again become popular, and that apoplexy might by this be often averted. Dr. Soper went on to speak of the practice of "spring medicine," of blistering, and of cupping, both wet and dry. All ointments were made on the premises in a huge mortar, with a pole and pestle attached to the ceiling; every tincture was home-made, and a powerful tincture press used. He passed in review many of the favourite prescriptions of the day, and stated his belief that time would show that natural products would hold their own against a vast number of synthetic compounds.

Therapeutical and Pharmacological Section.

January 28, 1908.

Dr. T. E. Burton Brown, C.I.E., President of the Section, in the Chair

Arterio-Sclerosis.

By W. E. Dixon, M.D.

It is well known that drugs when administered to animals under certain conditions may cause gross anatomical changes. Thus, if digitalis be administered to young pigs in moderate doses extending over a period of six weeks the heart hypertrophies, and post-mortem examinations have shown that such hearts may weigh twice as much as hearts obtained from normal animals of the same litter. The administration of drugs and toxins may give rise to certain arterial degenerations, and it is for this reason that I venture to draw the attention of the Therapeutical Section to a pathological subject.

Arterial disease in man may begin either in the intima (atheroma) or in the media, when the change is usually spoken of as arterio-sclerosis. Of the exact causation of these conditions we know little except that poisons, such for example as syphilis and lead, play a considerable part. Experimental arterial disease in animals has been produced in a great variety of ways. As early as 1889, Gilbert and Lyon showed that injections of bacteria and their toxins produced fibro-calcareous changes, especially if the vessel wall had been mechanically injured. Injury to the vessel has been shown by many to result in severe inflammatory conditions. Josué, in 1903, first drew attention to the action of drugs in producing arterial disease in animals. He injected adrenalin into the veins of rabbits and produced extensive changes in the middle coats of the arteries. In man it has often been shown that the first sign of this form of arterial disease is fatty degeneration, and Klotz has suggested that it is this fatty degeneration which is responsible for the attraction and deposition of calcium salts.

Within the last two or three years it has been shown by different workers in this laboratory that any drug which has the power of considerably raising blood-pressure will, when injected into the circulation of healthy animals, bring about degeneration of the middle coat of the arteries. Generally about six injections must be administered before any changes are observed, and the effects occur alike in young and middle-aged animals. It has been shown that digitalis, squill, apocynum, barium, lead, adrenalin, nicotine, and even the inhalation of tobacco smoke will all bring about these changes.

Incidentally, I might mention that the administration of thyroid gland given with these drugs does not appear to influence the ultimate effect on the vessels. It is well recognised that cretins and those suffering from myxœdema show early degenerative changes in their vessels, and hence has arisen the idea that arterial degeneration may be due to a deficiency in thyroid secretion, and it is with this idea that the iodides, drugs which are supposed to increase the activity of the thyroid gland, are so frequently administered in atheroma and allied conditions. Thyroid secretion, however, has nothing to do with the changes which we are considering here, whatever influence it may have on other forms of arterial degeneration.

Changes brought about in the vessels by any of the drugs mentioned are first rendered noticeable by granular degeneration in the muscle fibre. The muscle fibre losing its function, tone in the affected portion of the vessel is lost; the blood-pressure dilates this somewhat, and the elastic fibres lose their crenated form in the middle coat and appear as straight fibres. Considerable leucocytic activity can be observed at this time. Calcium salts are now deposited around the straightened elastic fibres, so that at this stage sections stained in silver nitrate and fixed in sodium hyposulphite show a black band in the substance of the media, whilst the intima may be either quite unaffected or appear a little thickened. The elastic fibres show a normal reaction to elastic stains and do not appear to be in any way affected. If these changes are induced in a more violent manner, such as by painting the vessels with irritants or by severe trauma, cartilage and bone may be formed in the vessel. These changes have been very definitely induced and demonstrated by Harvey.

The origin of these sclerotic changes produced by the administration of drugs has been put down (1) to a toxic effect and (2) to the simple mechanical rise in blood-pressure. Loeb and Githens injected into animals amyl nitrite along with adrenalin. They believed that the nitrite would entirely antagonise the blood-pressure raising properties of the adrenalin ; as the combined injection still caused arterial degeneration they concluded that adrenalin acted not by raising blood-pressure but by a toxic action. The essential premise of Loeb and Githens is, however, incorrect. Adrenalin and nitrite injected together do raise blood-pressure, although the rise is much shortened by the presence of the nitrite. If the toxic hypothesis is correct one would expect to obtain very severe degenerations by the injection of irritant drugs ; but this is not the case, for some of the essential oils and the cantharidates produce hardly any such effects. Lately we have obtained what I regard as very conclusive evidence that rise in blood-pressure alone can produce arterial disease. Dr. Harvey has shown that merely compressing the aorta of rabbits with the fingers for two or three minutes daily, thereby raising the blood-pressure 30 mm. or 40 mm. of mercury, produces degeneration of the aorta above the point of compression without causing any change in the vessel below. No other effect was observed on any tissue, and the rabbits appeared normal in every other way. The degenerative changes so induced were exactly comparable with those induced by the injection of drugs.

Three main hypotheses have been advanced to account for the initiation of these changes. Manchot suggested that breaking of the elastic fibres was the primary cause of arterio-sclerosis, and his idea has been supported by numerous other observers and does, no doubt, explain the patchy nature of the changes ; but in these experiments the elastic fibres are not found to be snapped across in the early stages of the disease but only in the later, after there has been considerable deposition of calcium salts. Moreover, if changes in blood-pressure such as we have described so readily cause breaking of elastic fibres, then it should be possible, on connecting a portion of the isolated rabbit's aorta with a mercury manometer, by alternately raising and lowering the pressure in this isolated vessel, to induce this change. Such a procedure, however, does not cause breaking of the elastic fibres.

Many writers have suggested that contraction, or changes in the vasa vasorum, may initiate the disease by giving rise to areas of anæmic necrosis, but in these experiments the vasa vasorum have never been observed as differing from those in normal vessels, and it appears to me highly problematical as to whether these vessels are capable of constricting under the influence of such drugs as adrenalin, because we know that the coronary arteries, which in a sense are of the same nature as the vasa vasorum, are unprovided with vasomotor nerves, and the blood-flow through them is entirely determined by the general blood-pressure.

All the evidence points to the muscle fibres being the essential seat of the disease. As these fibres are destroyed blood-pressure opens up the diseased portion of the vessel, the elastic fibres become stretched, calcium is deposited, and rupture of elastic fibres may occur.

In conclusion, I wish to express my views as the result of these experiments on two points. The first is on the question of exercise. We have found that violent dumb-bell exercise in men aged 60 raises the blood-pressure to twice the height or more, than similar exercise in men aged 23 or 24. The rise in pressure seems to be sudden in onset and to disappear very rapidly as soon as exercise is succeeded by rest. Tracings of these conditions I hope to publish on another occasion; all I would do now is to draw attention to the possibility of such considerable alterations in blood-pressure as those in the man aged 60 (30 mm. of mercury) being instrumental in the induction of arterial degeneration. The second point is on the question of smoking. We have abundant evidence to show that if a man unused to tobacco be induced to smoke a cigar, his blood-pressure first rises from 10 to 25 mm. of mercury, and after from fifteen to thirty minutes, if the smoking has been continued, drops suddenly from 30 to 50 mm. or more. The habitual and moderate smoker, however, if subjected to the same ordeal, shows no change beyond a slight rise of 4 or 5 mm. in his blood-pressure. In those who have acquired a tolerance to tobacco, smoking produces little effect on the blood-pressure, but in the non-tolerant sufficient alterations may occur, as has been demonstrated with rabbits, to induce a slight degree of arterial degeneration. In other words, the moderate and continuous smoker throws less strain on his blood-vessels than the man who smokes occasionally.

DISCUSSION.

Professor CUSHNY said he considered the paper to which they had listened was of extreme importance, and he hoped Dr. Dixon would continue his work. They must, of course, remember that in man the rise of pressure under digitalis was very much more gradual, and there was not the sudden jerk upon the vascular wall that there was in animals in experiments. He did not think there was such a serious rise in pressure in the case of man, except, perhaps, when it was due to very violent exercise.

Dr. T. D. SAVILL said he had listened with a great deal of pleasure to Dr. Dixon's paper. The subject of how arterial sclerosis was produced had interested him for a great many years. However, it struck him as a most extraordinary circumstance that so short an increase in blood-pressure as a few minutes daily should be able to produce so very marked an effect, and he thought it would be interesting to know a little more about the condition of the animals in the intervening periods. He would like to ask Dr. Dixon whether there was any other contributory cause that might have been in operation, such, for instance, as the age of the animals or their diet. It was an interesting fact that the principal changes took place in the middle coat. Dr. Savill had always held the view that the primary disease of the arteries began in the middle coat, and he had advanced reasons for this belief in a paper he gave before the Pathological Society of London.[1] There was never any disease in the other coats without changes in the tunica media also. Moreover, this tunic was the functionally active structure; the other tissues were simply limiting or supporting fabrics. He would also like to ask Dr. Dixon whether he noticed in the earlier stages the granular changes which took place in the muscular fibres. He hoped Dr. Dixon would be good enough to give them the benefit of his observations on the effects of nicotine. He thought that was an important matter, because, no doubt, many people hesitated as to whether they ought, or ought not, to give up the fragrant weed, and perhaps they even felt their pulses from time to time; probably Dr. Dixon's valuable researches would help them to come to a wise decision on the matter. In conclusion he wished to express his admiration for Dr. Dixon's work, which bore very directly on therapeutics. One of the most important questions that affected medical men in their daily practice was how arterial degeneration arose and how it could be prevented. "A man is as old as his arteries" was a quotation that was often before them, and if the investigation of this matter could be carried on in a laboratory so as to ascertain how and why arterial sclerosis came on, it would be difficult to measure its far-reaching results.

Mr. MABEN said he would like to ask Dr. Dixon if he controlled the results of his experiments by careful examination of supposedly normal animals obtained from the same sources and kept under the same conditions as those receiving the injections. He asked that question because it had recently been stated in the *Journal of the American Medical Association* by two workers that they had injected a large number of rabbits, and when they killed them they found undoubted signs of arterial sclerosis. But they also kept a number of rabbits of a similar character under the same conditions, and when these were killed they were found in almost the same proportions to show signs of arterial sclerosis. And those two workers had therefore come to the conclusion that possibly the disease may have actually been present in the animals before the injections.

The PRESIDENT (Dr. Burton Brown) asked Dr. Dixon if he would be good enough to favour the members with some remarks on nicotine.

Dr. DIXON, in replying, said he would like to point out that the work was not his own, and he did not wish to take any credit for the results; it was the work of three young medical men who had been working in Cambridge, particularly Dr. Harvey and Dr. Lee. The experiments had been carried out on all kinds of animals aged six weeks and upwards, and in only one case had he ever seen definite arterio-sclerosis in a control animal. It was not a question of whether there was slight arterial disease or not. In a certain number of cases there were no positive results, or only very slight results. With regard to the remarks of Professor Cushny, it should be remembered that when digitalis was given to men, it was not administered in the same way. The object in these experiments was to determine how arterio-sclerosis arose, and so they produced a large effect, that was to say, they produced a great strain in the blood-vessels. That was a condition that was not obtained in a man when digitalis was given. In answer to Dr. Savill he might say they always kept a number of controls of the animals. They all lived together in a very big case, ran about together and ate the same food. Turning to the question of nicotine, he said the one thing of importance was that it was comparatively easy to become immunised to nicotine. That immunisation was brought about by the production in the body of a ferment that destroyed the nicotine. We could all destroy a small amount of nicotine, but when an animal was immunised to nicotine a substance, probably a ferment, which destroyed nicotine could be obtained from the liver. If a boy who had never smoked inhaled a fairly strong cigar his blood-pressure would gradually go up to about 20 mm. of mercury or more, and would then drop very quickly, and might even go down as low as 50 mm. ; this would be associated with all the symptoms of collapse. If you experimented with an old smoker, a man who always had a pipe in his mouth, and let him smoke the same cigar his blood-pressure remained quite steady, while in the case of the moderate smoker the blood-pressure went up very little. Supposing it was 120, it would, perhaps, go up to 124 or 126, and would generally stay there for a considerable time, but there was no collapse afterwards. The point he wished to bring out was that it was much better to keep oneself more or less immune than to smoke occasionally a strong cigar.

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[1] *Trans. Path. Soc. Lond.*, 1904, lv., p. 375.

Dr. J. GRAY DUNCANSON said he was glad Dr. Dixon had ended with a word of consolation—that those who smoked might continue now to do so regularly. Somewhat the same results occurred with regard to athletics; it was the sudden severe exertion which told upon the circulation, and if repeated at irregular intervals might cause irreparable damage.

Nutmeg Poisoning.

By A. R. Cushny, M.D., F.R.S.

In most text-books on toxicology the oils of tansy, pennyroyal, and sage are mentioned as the members of the volatile oil group from which poisoning occurs, and I am not aware that any author has included nutmeg and its oils as a cause of symptoms. Yet a very considerable number of cases of poisoning with nutmeg are recorded in medical literature. Some years ago I suggested to Dr. G. B. Wallace, then my assistant, that he should collect these cases, and from a study of them he was induced to examine the pharmacological action in animals and to separate the poisonous constituent of the nutmeg. His results were published in 1903,[1] but as they are not very accessible I have thought it well to bring them before you.

Wallace found that cases of poisoning occurred exclusively from the use of the crude nutmeg or of mace, which, of course, is more readily accessible than most other poisons; in many instances it had been used as an abortifacient or emmenagogue, although in the cases of poisoning recorded it does not seem to have exercised any action on the uterus.

Curiously enough, the subject of nutmeg poisoning, which is ignored by present writers, was very early recognised by the older authorities on toxicology. Thus Lobelius, in 1576, relates a case in which a woman was rendered delirious by nutmegs, and Paullinus, in 1704, gives some experience in the matter. In recent years Wallace found twenty-five cases reported in the medical press, and there is every reason to believe that this represents only a small fraction of those occurring. Since Wallace's paper appeared, I have met two or three further instances of poisoning in the journals. The quantity of nutmeg used in these cases of poisoning varies, but a large number of patients stated that they had taken one or one and a half nutmegs grated or cut into fine pieces, so that this quantity appears to be sufficient to cause intoxication in many instances. One man is stated to have been poisoned by a teaspoonful of mace.

The symptoms generally come on in the course of one to six hours, and consist in drowsiness passing into stupor, from which the patient sometimes can be aroused with difficulty, and then is unable to recognise his surroundings, sees double, and gives other evidences of cerebral depression, although sometimes he may be able to answer questions. Delirium is frequently present before the stage of stupor, or the patient, on being aroused, may be delirious for a time. In some patients spasmodic movements of the jaw are recorded, in others continual laughter or persistent disconnected talk. Sometimes the first symptom is burning pain in the stomach, præcordial anxiety, or giddiness. In one or two cases an alarming degree of collapse occurred, and in others the pulse is noted to be small and thready, the feet cold, and the face pale. The pupils are generally widely dilated, and dryness of the throat may be felt at first. The stupor lasts for four to thirty hours and then passes off, and the patient recovers. Only one death from nutmeg is recorded—in the case of a boy, aged 8, who ate two nutmegs and passed into a comatose condition, from which he could not be aroused, and which terminated in death after about twenty hours. These symptoms present a similarity to those of opium, and more especially to those of cannabis indica, in which there is often the same mixture of depression and stimulation. The stupor and delirium point to some such mixed action on the brain as is met with under cannabis indica, and, in fact, few of the symptoms indicate the involvement of any other organ than the higher parts of the central nervous system. The pain and burning in the stomach arise from the irritant local action of the oil, and the symptoms of collapse may suggest some action on the circulatory centres, but they were not observed in the majority of cases.

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[1] "Contributions to Medical Research," dedicated to V. C. Vaughan, Ann Arbor, Michigan, 1903.

The nutmeg contains from 3 to 8 per cent. of volatile oil, and when this has been extracted from it the residue produces no effect whatever on animals, while small doses of the oil itself induce characteristic effects. The oil contains several terpenes and small quantities of higher boiling substances which can be separated by fractional distillation. The terpenes are devoid of action, except in enormous quantities, while the fraction boiling at 150° C. at 14 mm. pressure proved to be a powerful poison. The constituents of this highest fraction have been examined by several chemists, who have isolated bodies called myristicol (Wright) and myristicin (Semmler), and Wallace obtained a body which did not conform to the formulæ or properties of either of these, but which proved to be the active poisonous principle. He was inclined to consider his body closely related to myristicin (Semmler), and possibly derived from it, but the later investigations of Thoms and others indicate that Wallace's body was the true myristicin, while that described by

Semmler under this name was isomyristicin, formed from it by his manipulations and not occurring in the oil.

That nutmeg acts as a depressant on the central nervous system has long been recognised, and the older writers on materia medica—Murray,[1] Rumph, Pereira, Gmelin and others—describe its narcotic properties, and some of them even recommend it as a soporific. Experiments on animals have been performed by several investigators who used the volatile oil, but did not isolate the active ingredient in it.

Wallace, using the pure principle, found that frogs placed in a dilute watery solution of it at first showed some symptoms of restlessness (probably from the irritant action on the skin), but soon became depressed and finally completely paralysed, with abolition of the reflexes. The animal may recover from this if removed from the dish, but otherwise dies. The same effect is obtained when the drug is injected into the lymph sacs. The action is confined to the nervous system, the peripheral nerves and muscles being unaffected and the heart beating quite normally even when bathed in the oil. The rabbit reacts in a very similar way, showing symptoms of depression and finally of paralysis of the central nervous system, without any significant change in the circulation, even when the oil is injected intravenously. The animal passes into a condition of depressed reflexes, lessened movement and incoördination, which deepens into complete stupor ; sometimes tremor occurred or sudden twitches at intervals of some minutes. The cause of death is failure of respiration, the heart continuing to beat for some time afterwards. The cat is much more susceptible to the action than the rabbit, as is very generally the case with drugs acting on the central nervous system. About 0·4 G per kilo of the highest distillate given *per os* causes restlessness with weak spasmodic movements and tremor resembling that seen in carbolic acid poisoning, and profuse salivation. The restlessness passes into quiet with persistence of the tremor, incoördination of the movements, weak reflexes and partial anæsthesia. The pupils are dilated. Soon a stage of stupor, gradually deepening, sets in, the respiration is laboured and feeble and finally ceases some eight to twelve hours after the ingestion of the poison. In many cases, however, after some hours of stupor, a gradual improvement begins, and in fifteen hours from the taking of the poison the animal appears fairly normal save for unusual quietness and disinclination to move about. This improvement is only temporary, however, the cat again becoming weaker and more depressed, eating nothing and paying no attention to its surroundings, until coma returns followed by death in thirty-six to seventy-two hours from the time the oil was taken. Vomiting and purging are often seen. The blood-pressure remains unaffected even when large quantities of the oil are injected intravenously. When the oil is administered hypodermically the same symptoms follow, except that there is no salivation. If death is delayed symptoms of local irritation develop at the point of injection. The symptoms in the dog resemble those described in the cat.

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[1] This old Göttingen pharmacologist is perhaps one of the last believers in the tradition regarding the bird of paradise—"Avi quoque paradisiacæ in deliciis (nux myristica) est quæ vero inde inebriatur et mortua decidit, quibus factis formicæ adrepunt et pedes consumunt" (*Appar. Medicam*, 1792, vi., p. 138).

The symptoms in mammalia are thus, as in the frog, to be attributed to action on the central nervous system, which is depressed for the most part, but exhibits some indications of stimulation in the form of restlessness, slight convulsive movements, and tremor. Animals, therefore, correspond very closely to man in their reactions to nutmeg poison.

In addition, the oil exercises a marked local irritant action, whether given by the mouth or hypodermically. The wall of the stomach is found to be red and injected when it is given *per os*, and even more obvious signs of local reaction may often be met with in extravasations of blood and small areas of necrosis. The urine very often contains albumin along with some glycuronate. These effects in the stomach and kidney perhaps explain the secondary coma and death which follow apparent recovery in the cat. Many volatile oils induce fatty degeneration of the liver and other organs, but nutmeg poison has little or no action in this direction.

Wallace's results do not indicate any useful purpose which nutmeg might serve in therapeutics, but are of interest in drawing attention to the possibility of serious poisoning from one of our common domestic flavouring agents.

DISCUSSION.

Dr. F. B. POWER said he had been particularly pleased to hear Professor Cushny's paper on "Nutmeg Poisoning," and he was also pleased to have an opportunity of saying a few words on the subject. For several years past the subject had been one of considerable interest to him, not from the medical, but from the chemical standpoint; that was to determine, if possible, what were the constituents of nutmeg. He had observed in the journals numerous accounts of nutmeg poisoning from time to time, but nothing definite seemed to have been known respecting the cause of the poisoning. He was not aware whether Professor Cushny's work had been published. Some considerable time ago he (the speaker) decided to make a thorough investigation of the subject, and he had advanced considerably along the line. In the first place he obtained good Ceylon nutmegs, and having had all the volatile oils distilled from them, he subjected them to chemical investigation. He found that the essential oil of nutmeg was an exceedingly complex body, about which not much was known One or two of the constituent oils were known, but what struck him as peculiar was, he noticed that, when oil of nutmeg was injected into the circulation of a dog it produced pronounced narcotic effects that seemed somewhat different from those produced by nutmeg taken internally. In considering the character of the essential oil he found it to consist to the extent of about 90 per cent. of terpenes or bodies of the same composition as hydro-carbons, which were not practically regarded as narcotic, being particularly stimulants. In the essential oil of nutmeg he found no less than four alcohols. The points he particularly wished to bring forward for consideration were these: nutmeg poisoning was generally stated to be produced by one or two nutmegs, usually not more than two. The weight of two average nutmegs was about 10 grm. If they made a liberal allowance for the essential oil as being 10 per cent., then in two nutmegs weighing 10 grm. there would be 1 grm. of essential oil, and the question which he thought ought to be settled was, whether 1 grm. of the essential oil of nutmeg, when taken internally, would produce these effects. So far as he knew that had not been done. The effect was due to the high boiling constituents of the oil, although there was only a very small amount in the normal oil of nutmeg—about 4 per cent.: in 1 grm. of essential oil there would, therefore, be 4 cg. (about 0·6 gr.), or a little more than $\frac{1}{2}$ gr. in two nutmegs. Now the point was, would that amount of myristicin, when taken internally, produce the soporific action? He thought that point ought to be decided. In the course of his work he had looked up a number of papers on the subject, and he found some work had been carried out in Kobert's laboratory about four years ago by one of his assistants, who especially investigated the action of myristicin. But those experiments were all conducted with small animals, such as rabbits and fowls, rabbits chiefly. The experimenter injected from 1 grm. to 2 grm. of pure myristicin into the circulation, and, of course, that produced death. That 1 gm. of myristicin would represent about twenty-five times as much as was contained in two nutmegs, and yet that was given to an animal weighing about 2 lb., therefore it would seem that the effect of the substance when administered in that way could hardly be compared with the effects produced when it was taken through the mouth. It was also evident that the action upon animals was very much less than in the case of man. At the present time he was engaged in investigating the other constituents of the nutmeg. They thought perhaps some other toxic principle might be found in the nutmeg itself. That idea seemed to be borne out by some reports published by an American physician, who some years ago stated that he had been called in to a great many cases of nutmeg poisoning. He said sometimes children ate them without knowing the effects, and the effects were very similar to those of an overdose of chloral. Much of the essential oil contained very little myristicin. The "British Pharmacopœia," unfortunately, prescribed certain requirements for the oil of nutmeg with regard to specific gravity which really excluded some of the more important aromatic parts of the oil, including some myristicin. Such an oil might have been administered, and hence the difference in action. He could not yet say very much about the other constituents of the nutmeg. The fatty oil would naturally not be expected to contain this principle: it contained the glycerides and ordinary fatty acids. They, however, were examining very carefully the material from which the fatty oil of nutmeg was expressed. They had had large quantities of it, and, of course, that contained everything except the fatty and essential oils; it contained a number of principles of a resinous nature which had not yet been tested, and he did not know whether they would find anything active in it: but he thought it would be very interesting and very important that they should be able to definitely decide as to the cause of the poisoning, and that could best be accomplished by using the essential oils and administering them in such amounts as were actually contained in the nutmeg. And, if they found it required such a large amount to produce the effect, it could hardly be due, it seemed to him, to these bodies.

Dr. J. GRAY DUNCANSON said he thought they had all been pleased to hear so much about nutmeg. There were various varieties of nutmeg, and these were now grown both in Asia and America, the Penang being the best. Some people were very susceptible to nutmeg, and even a small quantity grated on the top of a pudding made them sick. He would like to direct Professor Cushny's attention to the fact that nutmeg poisoning had not been quite so much neglected in English medical literature as he had led them to believe. A week or two ago he (the speaker) had occasion to look up Taylor's "Medical Jurisprudence," and, knowing this paper was going to be given, he read the article on nutmeg poisoning and he found Taylor cited two or three cases; the symptoms in those cases seemed to be of two kinds, cerebral and gastro-intestinal hallucinations and stupor. He had only met with one case of nutmeg poisoning himself, and that was a pregnant woman who had taken the whole of a nutmeg grated up in gin. She certainly aborted, but whether this was entirely due to the nutmeg or to some other mixture she had been taking at the same time he did not know. She had severe symptoms of the character already described, and ultimately made a good recovery. In India the oils were obtained from the nutmeg by pressure and then used as a lotion for the hair, said to have stimulating and anti-parasitical properties.

Professor CUSHNY, in reply to Dr. Jackson, who asked whether it were possible that the narcotic and stimulating principles were one and the same, said he had no reason to suppose that they were different substances. He might also mention that they had considered the question of the large dose comparatively that was necessary to poison animals, and they found it required a much larger dose to narcotise a dog with other bodies, such as cannabis indica, than was necessary to induce symptoms in man.

Therapeutical and Pharmacological Section.

February 25, 1908.

Dr. T. E. Burton Brown, C.I.E., President of the Section, in the Chair.

The Therapeutics of Diet.

(An Address given at the Annual Conversazione.)

By Harry Campbell, M.D.

I will first ask your attention to a diagram, which represents what may be termed the evolution ladder; that is to say, the ladder up which the evolving man has mounted from the simian or ape. In this ladder I have put fifteen rungs. Man is derived from a species of ape, but not from one exactly like any of the existing apes. He and they are derived from a common ancestor, which I place on the third rung, the existing anthropoid apes being on the fifth rung, and the average European on the fifteenth. Each successive rung indicates one additional unit of brain substance. Such a ladder affords an accurate means of representing the evolution grades through which man has passed in his ascent from an ape-like being. Perhaps some day we may have a twentieth-rung man. I propose to draw your attention to three great advances which have been made in the diet of the evolving man. There can be no doubt that our simian ancestor was frugivorous; that is, he subsisted for the most part on a condensed form of vegetable food. By a herbivorous animal we mean one which, like the horse or the cow, subsists on a bulky form of vegetable food; the frugivorous animal, on the other hand, selects more condensed and nutritious substances, such as nuts and berries. The existing anthropoid apes, in a state of nature, also include in their dietary a certain amount of animal food, such as eggs, fledglings, small reptiles, insects and grubs. The first great dietetic advance made by man was at about the tenth stage of his ascent: it consisted in the employment of artificial means for procuring his food.

When man learned to hunt, to stalk and to trap animals, and to fish, you will see at once that he was able to add very largely to his dietary. From the time he began to do this to the next advance we can speak of as the early hunting period. This next advance was made when man began, say at the twelfth stage, to employ artificial means in the preparation of his food. I have no doubt that they were, in the first instance, employed solely in regard to vegetable food. The first artificial process to which man subjected his food was probably that of pounding or grinding it. The object of this was to break up the dense meshwork of cellulose in which the "foodstuffs"—starches, sugars, albumins and fats—are entangled. Vegetable tissue consists of a series of minute chambers, the walls of which are made of cellulose. This cellulose is absolutely non-digestible by man. Consequently, in order that he may get the nutritive material out of raw, unprepared vegetable tissue, it is necessary for him, either by mastication or other means, to break up the cellulose. It is for this reason that all the vegetable-feeding mammals subject their vegetable food to prolonged mastication. This is well shown in the case of the cow chewing the cud. This animal swallows the grass on which it feeds rapidly, and then, at its leisure, regurgitates it and subjects it to a prolonged grinding action so as to break up the dense cellulose and liberate the contained nutriment.

Later, man learnt to cook his vegetable food, the effect of cookery being the same as that of pounding; when vegetable tissue is cooked, the starch granules swell up and rupture the cellulose chambers.

The third great advance, which I place at about the thirteenth rung, consisted in the artificial production of food in the breeding of animals and in the cultivation of plants. This enabled man to take a tremendous stride forward, because before he adopted food culture practically the whole of his time was taken up with the food quest. When a certain section of the community undertook the supply of food the remaining portion could turn its attention to other things, and civilisation became possible by the division of labour. Here let me observe that a given acreage of land can produce more vegetable than animal food, and consequently the total effect of food culture, or cibiculture, as we may term it, has been to increase the vegetable more than the animal food.

I would now like to draw your attention to a second diagram, which represents the probable relative quantities of animal and vegetable food which man has consumed during the various phases of his evolution from the simian. Starting at the fifth rung, when the simian began to develop into the homosimian, you will see that the animal food was very small in proportion to the vegetable, but as the evolving man grew in intelligence he was no longer content with a small quantity of vegetable food. He used his intelligence to procure the much more nutritious and more highly prized animal food, and hence with his mental advance he became more and more carnivorous, less and less vegetarian. When he devised means of stalking and trapping animals and of fishing he became still more carnivorous, and, at the end of the early hunting stage, was, I believe, actually more carnivorous than vegetarian. When he began to cook his food he was enabled to increase his supply of vegetable food, and so from the beginning of the cookery period began to subsist less and less on animal food, though right up to the agricultural period he continued to take large quantities of animal food. Thus the primitive Australians, who are in the early cookery period (*i.e.*, they cook their food, but do not breed animals or cultivate the soil), live on about equal parts of animal and vegetable food, and the like is true of most other extant pre-cibiculturists. With the last stage of all—the cibicultural—when the vegetable food was increased more than the animal food, man continued to become less carnivorous, and at the present moment he consumes, on the average, about two-thirds vegetable to one-third animal food.

We may conclude that man has evolved from the ape to be "the roof and crown of things" on a diet which is largely carnivorous, and if such is the case it is obviously idle to contend that animal food is necessarily harmful to him. There are some who thrive better on a diet which is largely vegetarian than on an animal diet, but these people really have no reason to be proud of themselves—they are subnormal, not improbably reversions to a more primitive order of things. I do not mean to say that when we get to the twentieth rung of the ladder the amount of animal food consumed by the super-man may not have reached the vanishing point. This is a consummation devoutly to be wished, because the consumption of animal food has many gruesome accompaniments, but it is certain that as at present constituted man is a mixed feeder.

I now propose to trace, very briefly it must be, some of the changes which have taken place in man's vegetable food since simian times. As the evolving man was by his increasing intelligence able to procure more and more animal food, he was able to exercise a greater choice in the selection of his vegetable food and thus to abandon the coarser varieties. When he reached the stage of cookery his consumption of raw vegetable food underwent considerable diminution and has steadily diminished until the present time, when very little vegetable food is consumed in the raw state, and this for the most part of the cultivated kind which can be swallowed after comparatively little preliminary mastication. Furthermore, the cooked vegetable food has during the entire agricultural period been tending to get softer, so that at the present time not only does man consume little or no coarse vegetable food in the raw state, but most of his cooked vegetable food is of a kind which does not compel vigorous mastication. This is not only true of so-called "vegetables," such as potatoes and greens, but of almost all of the present day farinaceous foods, which consist of refined bread (from which the crusts are often carefully removed), pudding, porridge, cakes, buns, muffins, scones, blanc-mange and the like. In other words, almost the sole vegetable food we now consume is of a soft, spongy or pappy kind, so much so that we might almost call this the age of pap. Until man discovered the art of preparing his vegetable food he was compelled, like any vegetable feeding mammal, to grind it laboriously with his teeth, so as to break up the cellulose network. In so doing, the starch was intimately mixed with the saliva and largely digested in the mouth, while the jaws and teeth had abundant work to do. But at the present time vegetable food, being soft and easily swallowed, passes down into the stomach without being properly masticated and insalivated, and in consequence of this the stomach tends to be flooded with a superabundance of crude starch, while the jaws and teeth do not get the exercise they crave for. Hence, on the one hand, we get dyspepsia and its many evil consequences, and on the other hand, defective jaws and teeth. I am convinced that an untold amount of disease could be prevented if we would only take the precaution to consume a due proportion of our vegetable food in a form which compels abundant mastication. I am convinced that by following out the simple rule of giving children from early years a due proportion of hard vegetable food, five-sixths of the dental diseases among us would be obviated, and that the frequency of diseases of the nose and throat would be much reduced.

Lastly, in regard to sugar. Before man cultivated the vegetable kingdom, he was dependent for his sugar upon that contained in fruits and other vegetable substances, and also upon wild honey. With the advent of the agricultural period and the cultivation of such highly sugary vegetable foods as the date, the fig, the sugar-cane and beetroot, and, above all, when he came to extract pure sugar from these latter substances, the supply of sugar was enormously increased; at the present time thousands of tons are annually extracted. Now, I do not say that sugar is not, in moderation, a good food, but I would draw your attention to the danger of consuming too much of it.

Therapeutical and Pharmacological Section.

March 24, 1908.

Dr. T. E. Burton Brown, C.I.E., President of the Section, in the Chair.

Hypnotism.

By J. Milne Bramwell, M.B.

Hypnotism is too wide a subject to be condensed into a single lecture, and I do not mean to attempt the feat. I simply propose to talk to you in a somewhat informal manner; to give you an outline sketch of its origin and development; and, finally, to say something about my own work.

Hypnotism is popularly regarded as of French extraction, and few seem to be aware that England was its birthplace. To understand its origin and development we must go back to the later mesmerists.

John Elliotson.

About seventy years ago John Elliotson, the foremost physician of his day, had his attention attracted to certain mesmeric experiments and commenced to investigate the subject on his own account. He succeeded in mesmerising a number of patients in University College Hospital, and thus cured or relieved many forms of nervous disease, although at that time the principle of suggestion was not understood as it is now. In Elliotson's day the phenomena were supposed to be due to a mysterious force or fluid, which was transmitted from the operator to the patient. This force, it was also thought, could be passed into magnets, metals and other inanimate objects, which in their turn were supposed to be capable of exciting mesmeric phenomena.

Elliotson's work soon attracted attention, and students from other hospitals flocked to his demonstrations in such numbers that he had to exchange his lecture room for the large operating theatre. His colleagues, however, persecuted him in many petty and disgraceful ways, and, while boasting that they would not even look at his demonstrations, they at the same time denied their utility. After a time, an order was passed by the council of the hospital forbidding mesmeric work within its walls, and Elliotson at once resigned all his hospital appointments. From his work there originated, however, a great mesmeric movement in England, and numerous painless operations were reported. As neither chloroform nor ether had then been discovered, mesmerism was the first anæsthetic, and one would have thought that even one or two successful mesmeric operations would have excited the benevolent interest of the whole profession. The contrary was the case; one surgeon performed 200 painless mesmeric operations, but they were never even reported in the medical journals; indeed, mesmerism was only referred to in order to heap insult and abuse upon all those connected with it. For example, in Nottinghamshire, in 1842, Mr. Ward, surgeon, amputated a thigh during mesmeric trance; the patient lay perfectly calm during the whole operation, and not a muscle was seen to twitch. The case, reported to the Royal Medical and Chirurgical Society, was badly received. Dr. Copland proposed that no account of such a paper having been read before the Society should be entered in its minutes. He asserted that if the history of the man experiencing no agony during the operation were true, the fact was unworthy of their consideration, because "pain was a wise provision of Nature, and patients ought to suffer pain while their surgeons were operating; they were all the better for it and recovered better." Elliotson was practically ruined through his mesmeric researches and work, and died a broken-hearted man.

JAMES ESDAILE.

James Esdaile was born at Montrose in 1808. He studied medicine at Edinburgh University, graduated there as M.D. in 1830; obtained a medical appointment in the service of the East India Company, and arrived at Calcutta in July, 1831.

Esdaile made his first mesmeric experiment in 1845 when in charge of the native hospital at Hooghly. The subject was a Hindoo convict who had just undergone a painful surgical operation, which was about to be repeated. At this time Esdaile knew nothing of mesmerism, except what he had read in the daily papers, but it occurred to him to try to mesmerise this patient in order to render him insensible to pain. The experiment was successful, and was again repeated on the same subject a week later. This encouraged Esdaile to persevere, and at the end of a year he reported 120 painless operations to the Government. As up to this time no anæsthetic of any kind had been employed in surgical operations in India, his cases naturally excited much attention. A committee, largely composed of medical men, was appointed to investigate his work; their report was favourable, and Esdaile was placed at the head of a Government Hospital in Calcutta for the express purpose of mesmeric practice. From this date, until he left India in 1851, he occupied similar posts. He recorded 261 painless capital operations and many thousand minor ones, and reduced the mortality in the removal of the enormous tumours of elephantiasis from 50 to 5 per cent. Patients flocked to him from all parts of the country, and the record of his painless mesmeric operations forms one of the most fascinating and romantic pages in the whole history of medical science.

After leaving India, in 1851, Esdaile settled in Perth, and about a year later informed Elliotson that he had found the inhabitants of the far North as susceptible to mesmerism as those of the farthest East. Dr. Fraser Thompson, physician to the Perth infirmary, became a convert and performed some successful mesmeric operations. His colleagues, however, called a meeting of the directors, and stated that they would resign if the practice of mesmerism were permitted in the hospital.

JAMES BRAID, THE FATHER OF HYPNOTISM.

James Braid commenced to investigate the subject of mesmerism in 1841. He was born in Fifeshire in 1795, had studied at Edinburgh University, and at this time was in practice in Manchester, where he had already gained a high reputation as a skilful surgeon. Braid believed mesmeric phenomena were due to self-deception or trickery, and at the first mesmeric séance at which he assisted saw nothing to cause him to alter his views. Six days later he noticed that one subject was unable to open his eyes. He regarded this as a real phenomenon and was anxious to discover its physiological cause; and the following evening, when the case was again operated on, he believed he had done so. After making a series of experiments, chiefly on personal friends and relatives, he expressed his conviction that the phenomena he had witnessed were purely subjective, and commenced almost immediately to place these views before the public. In 1843 Braid published "Neurypnology, or the Rationale of Nervous Sleep." This was followed by many other works of more or less importance, and of these I have been able to trace forty-one, but all have long been out of print in this country.

According to Braid, the phenomena of mesmerism depended entirely on the physical and psychical condition of the patient, and were absolutely independent of the volition of the operator or of any mystical or magnetic fluid which emanated from him. From the physiological side he explained the phenomena by changes in the nervous system of the subject. These consisted in the exhaustion of certain nerve centres, with resulting decrease in the functional activity of the central nervous system; they arose from continued monotonous stimulation of other nerves, *e.g.*, those of the eye, by fixed gazing; those of the skin by passes with contact. He explained the phenomena psychologically by concentration of attention and monoideism. The mind was so engrossed with a single idea as to render it dead to all other influences; the attention was concentrated upon the particular function called into action, while the others passed into a state of torpor. Only one function was active at any one time, and hence intensely so; the arousing of any dormant function was equivalent to superseding the one in action.

Braid proposed to substitute the term "hypnotism" for that of "mesmerism," and invented the general terminology of the subject, which remains little altered to the present day. He performed many experiments in order to test the alleged powers of magnets, metals, drugs in sealed tubes, &c., and found that the phenomena described by the mesmerists appeared when the patients had preconceived ideas on the subject or when these were excited by leading questions, but were invariably absent when they were ignorant of what was being done. Real magnets had no effect when the patients were unaware of their presence, while pretended magnets produced the phenomena when the patients knew what was expected to occur; and thus the mind of the patient alone was sufficient to produce the effects attributed to magnetic or odyllic force. Many cases of alleged clairvoyance and thought transference were also investigated by Braid, but he was never able to find anything but hypnotic exaggeration of natural powers.

Dangers.—According to Braid the hypnotic subject acquired new and varied powers, but did not at the same time lose his volition or moral sense. During hypnosis the patients evinced great docility, but were quite as fastidious of correct conduct as when in the natural state; they would neither reveal secrets nor accept improper suggestions. Braid stated that he had proved that no one could be affected by hypnotism at any stage unless by voluntary compliance. Hypnosis, in Braid's opinion, was not necessarily associated with loss of consciousness, and in many of his most successful cases the patients were afterwards able to recall all that had taken place. He claimed that he could hypnotize his patients more quickly than the mesmerists could influence theirs, and also that his curative results were superior, despite the fact that he neither believed in, nor invoked, occult powers.

In 1859 Dr. Azam, of Bordeaux, became acquainted with Braid's hypnotic work and commenced to investigate the subject for himself; an account of his experiments, with much reference to Braid, appeared in the *Archives de Médecine* in 1860. From this date the subject of hypnotism was never lost sight of in France, but it was not until forty years after its original publication that "Neurypnology" was translated by Dr. Jules Simon.

At Braid's death, in 1860, hypnotic work practically ceased in England, despite the attention drawn to it by Professors Carpenter and John Hughes Bennett. As we have just seen, however, the torch that Braid had lighted passed into France.

Dr. A. A. Liébeault.

Liébeault was born in 1823, and commenced to study medicine in 1844. In 1848 he read a book on animal magnetism; this impressed him greatly, and a few days later he successfully mesmerized several persons. He received his M.D. in 1850, and shortly afterwards started country practice. He worked hard and was often in the saddle making his rounds at 2 a.m. He had no private fortune, but in ten years he saved enough to enable him to live independently of his profession. In 1860 he began to study mesmerism seriously, just at the time that Velpeau communicated Azam's experiments to the Académie de Médecine. In order to find subjects for experiment Liébeault took advantage of the parsimonious character of the French peasant. His patients had absolute confidence in him, but they had been accustomed to be treated in the ordinary manner. He therefore said to them: "If you wish me to treat you with drugs I will do so, but you will have to pay me as formerly. On the other hand, if you will allow me to hypnotize you I will do it for nothing." He soon had so many patients that he was unable to find time for necessary repose or study. In 1864 he settled in Nancy, lived quietly on the interest of his capital, and practised hypnotism gratuitously among the poor.

For two years he worked hard at his book, "Du Sommeil et des États analogues, considérés surtout au point de vue de l'action de la Morale sur le Physique," but of this one copy alone was sold. His colleagues regarded him as a madman, the poor as their providence, calling him "the good father Liébeault." His clinique was crowded with patients; of these he cured many who had vainly sought help elsewhere, and few left him without having received benefit. In 1882, Liébeault cured an obstinate case of sciatica of six years duration, which Bernheim had treated in vain for six months. In consequence of this, Bernheim visited Liébeault. This was a great event in the life of the humble doctor. At first Bernheim was sceptical and incredulous, but soon this changed into admiration. He multiplied his visits and became a zealous pupil and true friend of Liébeault. In 1884, Bernheim published the first part of his book, "De la Suggestion," which he completed in June, 1886, by a second part entitled "La Thérapeutique suggestive." From this date, Liébeault's name became known throughout the world. The first edition of his book was quickly bought up, and doctors flocked from all countries to study the new therapeutic method.

Braid anticipated many of the most important observations of the School of Nancy, but we ought not, on that account, to undervalue the services of that school, and more especially those of its founder—Liébeault. Braid's researches were undoubtedly the exciting cause of the hypnotic revival in France, but little or nothing was known of any of his works except "Neurypnology," and his last MS., which contained some of his later views, was not not published in France until 1883. Liébeault independently arrived at the conclusion that the phenomena of hypnotism were purely subjective in their origin, and to him we owe the development of modern hypnotism.

Another point in reference to their careers is worthy of note. Braid's views at once brought him fame. His books sold rapidly, the demand for them exceeding his power of supply. The medical journals were open to him to an extent which may well excite envy in those interested in the subject at the present day. Liébeault's book, on the contrary, remained unsold; his statements only found sceptics, his methods of treatment were rejected without examination, and he was laughed at and despised by all. From the day he settled in Nancy in 1864, until Bernheim, some twenty years later, was the means of bringing him into notice, Liébeault devoted himself entirely to the poor, and refused to accept a fee lest he should be regarded as attempting to make money by unrecognized methods. Even in his later days, fortune never came to him, nor did he seek it; and his services—services which he himself with true modesty described as the contribution of a single brick to the edifice many were trying to build—only began to be appreciated when old age compelled him to retire from active work. Though his researches have been recognised, it is certain that they have not been estimated at their true value, and that members of a younger generation have reaped the reward which his devotion of a lifetime failed to obtain.

CHARCOT, OR THE SCHOOL OF THE SALPÊTRIÈRE.

While Liébeault's work may with justice be regarded as a direct continuation of that of Braid, there exists little or no difference between the theories of Charcot and that of the later mesmerists. The views contained in Burg's "Metallo-Therapia," which had been translated by Elliotson, were again adopted at the Salpêtrière. All the old errors, the result of ignoring mental influences, were once more revived. Medicines were again alleged to exercise an influence from within sealed tubes, the physical and mental conditions of one subject were stated to be transferable to another, or even to an inanimate object. In these views, and their refutation by the School of Nancy, we have an exact counterpart of the old controversy between Braid and the mesmerists.

The chief apostle of these doctrines was Luys, and considerable attention was drawn to him in this country in 1893 by popular articles in the daily papers and elsewhere. Indeed, the late Mr. Ernest Hart thought them of sufficient importance to demand his writing a book in order to disprove them. He, apparently, was ignorant of the fact that M. Dujardin-Beaumetz had, in 1888, reported to the Académie de Médecine that Luys' experiments were conducted so carelessly as to rob them of all value, and that amongst students of hypnotism they were entirely disregarded.

Although the theories of Charcot were practically identical with those of Elliotson, there is a point of contrast between them which must not be forgotten. Elliotson and Esdaile, however mistaken in their theories, were far in advance of their fellows. Amid much that was false they had discovered genuine phenomena and investigated them in a scientific spirit, and successfully employed their knowledge for the relief of pain and the cure of disease. On the other hand, Charcot's revival of old mesmeric fallacies did much to discredit hypnotism and to prevent its advance.

Coming now to more recent times, I propose to tell you something about my own work. As I have already mentioned, when Esdaile left India he settled in Perth. There my father, the late Dr. James Paton Bramwell, was in practice. He witnessed many of Esdaile's experiments, and he himself repeated them at a later date. These I saw when a boy, and later, when at college in Edinburgh, my attention was again drawn to the subject by Professor John Hughes Bennett, who gave a lecture every year on hypnotism. He begged us to keep our eyes upon the subject, which he predicted would one day revolutionize medicine. It has not done so yet, however, but I always found consolation in the earlier days of my work, when hypnotism was not recognized as a quite orthodox method of treatment, that, at all events, I was following the teaching of my own University.

Shortly after qualifying, I became engaged in busy general practice, and hypnotism was forgotten for the time being. One day, however, I was called upon to treat a case of *grande hystérie*, and I could find nothing about the disease in the books I then possessed. In a French work which I purchased I found not only an account of the disease, but also of its treatment by hypnotism, and I determined that one day I would go to France to see this for myself. Before this opportunity arose, however, I met with a case in my own practice which appeared to be suitable for suggestion, and of which the following is the history:—

Mr. —, aged 24, consulted me in May, 1889. Some months previously he had had a number of diseased glands removed from the face and neck, and went to the Mediterranean to recruit. While crossing a plank he fell and injured his perineum; an abscess formed, which burst externally and into the urethra. When I saw him there was a large unhealthy wound through which the urine escaped. I instructed him to pass a soft catheter regularly, and the wound became more healthy. One day he was impelled to empty his bladder before he could pass the instrument, and the water again escaped from the wound. This happened more and more frequently; at last the idea of passing water caused him at once to empty his bladder, no matter where he was at the time. This appeared to be entirely independent of the physical condition of the bladder, which did not contract because it was full or uncomfortable, but because the idea of urination presented itself to the patient's mind and was instantly translated into its physical equivalent. He now thought constantly about his condition, which thus became greatly aggravated. He began to sleep badly, and awoke frequently during the night; the instant he did so he thought of his bladder and was immediately compelled to empty it. Despite treatment, these symptoms continued for several months, and his state became a grave one. I had not previously employed hypnotism, but the mental element in this case was so marked that I determined, since other treatment had failed, to try what this would do. After explaining to my patient, an educated man, that I had no practical, and only slight theoretical, knowledge of the subject, I proceeded to hypnotize him by Braid's method. In a few minutes his eyeballs rolled upwards and inwards, and he became lethargic. I repeated this the two following days, then suggested during hypnosis that he should cease to think of his bladder, should always be able to pass his catheter, retain his urine eight hours, and sleep well. These suggestions were immediately fulfilled; from that day there was no return of his troublesome symptoms, and the wound healed without operation in about twelve months. At the present date (March, 1908) Mr. — is in good health.

Shortly after this I began to find that I could frequently induce insensibility to pain by suggestion, and I commenced to use hypnotic anæsthesia in minor operations. I happened to mention this to a dental friend at Leeds—the late Mr. Arthur Turner. He was interested, but incredulous, and I invited him to come to Goole to perform a number of dental operations under hypnotic anæsthesia. I was able to give him a large choice of patients, and he selected those cases which he considered would most severely try the method adopted. He painlessly extracted forty teeth, and published an account of his observations in the *Journal of the British Dental Association* for March 15, 1890.

His article attracted attention, and I was shortly afterwards asked to give a demonstration of hypnotic anæsthesia at Leeds. This I did on March 28, 1890, when upwards of sixty medical men and dental surgeons were present. The following amongst other operations were performed. In the first instance I am about to quote I was not present during the operation. I remained in an adjoining room, and sent the patient the written suggestion that she should go to sleep and obey the commands of Mr. Turner, the dentist. Immediately on reading the note she passed into a condition of deep hypnosis, during which sixteen teeth were extracted. She did everything Mr. Turner suggested, rinsed her mouth, &c. No gag was used. It was observed that there was a diminished flow of saliva and that the corneal reflexes were absent, the breathing was more noisy than ordinary, and the pulse slower. In another case, a boy aged 8, I had only attempted to induce hypnosis for the first time two days before the operation. Mr. Mayo Robson performed evulsion of the great toe-nail and removed a bony growth and part of the first phalanx. At the conclusion of the operations Mr. Pridgin Teale stated "that the experiments were deeply interesting and had been marvellously successful." The after-condition of the patients was remarkable; none of them suffered any pain, and the unpleasant symptoms which sometimes follow the use of anæsthetics were absent. They all made a hearty meal, and then returned to Goole, a journey of over an hour by train. The nurse in charge told me that she might have been conducting a party home from a fair, as they passed the time in laughing and singing. An independent account of the operations is to be found in the *Lancet* of April 5, 1890, p. 771.

Unfortunately the uncertainty of the induction of hypnotic anæsthesia renders it of little practical value, and I should never dream of wasting time in attempting to induce it, seeing that we possess other and more reliable anæsthetics. It is, however, of great scientific interest. It is not the mere anæsthetization of some particular group of nerve endings, such as cocaine produces, nor is it such an anæsthesia as accompanies the profound unconsciousness produced by chloroform and ether. On the contrary, by hypnotic suggestion you can select and inhibit, from amongst the percipient's possible sensations, disagreeable ones alone. For example, analgesia only can be induced, and the patient, apparently wide awake, may talk with the surgeon and his assistants during the operation.

At the same time that I was using hypnotism as an anæsthetic, I was also employing it still more largely in medical cases selected from amongst the patients in my own general practice. I do not propose to inflict upon you a detailed account of these, but I succeeded in relieving or curing many cases of functional nervous disease which had resisted all other forms of treatment. Amongst the more interesting was one of hyperidrosis in a girl, aged 15. On the back of the left forearm, a patch of skin, about 2½ in. long by 1½ in. broad, was the seat of constant perspiration. This condition, which had existed from infancy, was always excessive, and invariably rendered more so by emotion or exertion. The forearm was always enveloped in bandages, but these rapidly became saturated, and then the perspiration dripped upon the floor. The patient was easily hypnotized at the first attempt, and the following day the perspiration had markedly diminished. Hypnosis was again induced, when the perspiration entirely ceased; no relapse.

Immediately after the report in the *Lancet* of the demonstration at Leeds, medical men began to send me patients from different parts of the country. These were nearly all cases of long-standing nervous disorder, in which many other forms of treatment had been tried without success. They proved much more difficult than the patients drawn from my own practice, and sometimes I spent weeks before I was able to induce the slightest hypnosis. I was so disappointed with my results that I determined to try and find out if it were possible to improve my methods. I then visited all the more important hypnotic cliniques in France, Germany, Belgium, Holland, Switzerland and Sweden, sometimes spending weeks at a time at one clinique alone.

One of the most important things I learnt was that deep hypnosis was not essential in order to cure disease by suggestion. This view, which Bernheim particularly emphasized, I discovered later in a pamphlet of Braid's. In it he stated that he had found that only one in ten of those he cured by suggestion passed into any condition which even superficially resembled sleep. He proposed, therefore, to abolish all his own terminology, as he found that it was extremely misleading and caused patients to believe that they could not be cured unless they had been put to sleep.

The following is now my usual method: I rarely attempt to induce hypnosis the first time I see a patient, but confine myself to making his acquaintance, hearing his own account of his case, and ascertaining his mental attitude with regard to hypnotism. I usually find, from the failure of other methods of treatment, that the patient is more or less sceptical as to the chance of his being benefited. In most cases, also, he has either read misleading sensational articles on hypnotism or his friends have painted its dangers in striking colours. I endeavour to remove erroneous ideas, and refuse to attempt to induce hypnosis until the patient is satisfied of the safety and desirability of the experiment. I never tell a patient that I am certain of being able to hypnotize him, but always explain how much depends upon his own mental condition and power of carrying out my directions.

I further explain to the patient that presently I shall ask him to close his eyes, and then begin to make suggestions. I tell him that the important point is that he should concentrate his attention upon some drowsy mental picture, and try to turn it away from me. While he is doing this I make suggestions of two kinds. The first are in reference to the condition I wish to induce while he is actually in the armchair. Thus, I suggest: "Each time you come to see me, you will find it easier to concentrate your attention upon something restful; you will become more and more drowsy and lethargic," &c., &c. The other suggestions are the curative ones, and these vary according to the nature of each individual case. I explain to the patient that I do not expect these suggestions to be responded to at once, though this does occur in rare instances, but that it is the repetition of the suggestion, made in this particular way, which brings about the result. Thus, from the very first treatment the patient is subjected to two distinct processes, the object of the one being to induce hypnosis, that of the other to cure or relieve disease; and frequently the latter is successful before the patient can be described as genuinely hypnotized.

These curative suggestions apparently resemble those sometimes made in ordinary life by medical men and others. The results, however, are often very different. For example, a patient suffering from dipsomania had received many and varied suggestions. The loss of health, fortune and friends were powerful suggestions to stop drinking. Twelve months passed voluntarily in a retreat ought to have also had a strong suggestive influence. They all failed, however, while treatment by suggestion, associated with hypnotic methods, was speedily followed by success, and the patient has now been for many years an abstainer.

It is difficult to estimate the exact value of hypnotism in comparison with other forms of treatment. There are, however, one or two broad facts that ought to be kept in mind:—

(1) Hypnotism is not a universal remedy. As Braid truly said: "Whoever talked of a 'universal remedy' was either a fool or a knave." It is simply a branch of medicine, and those who practise it sometimes combine it with other methods of treatment. Thus, in some instances it is difficult to say what proportion of the curative results are due to hypnotism and what to other remedies.

(2) On the other hand, many cases of functional nervous disorder have recovered under hypnotic treatment after the continued failure of other methods. Further, the diseases which frequently respond to hypnotic treatment are often those in which drugs are of little or no avail. For example, what medicine would one prescribe for a man in physical health who had suddenly become the prey of an obsession? Such patients are rarely insane; they recognize that the idea which torments them is morbid; they can trace its origin and development, but yet they are powerless to get rid of it.

(3) In estimating hypnotic results, it must not be forgotten, too, that the majority of cases treated in this way are extremely unfavourable ones. As the value of hypnotic treatment and its freedom from danger become more fully recognized, it will doubtless be employed in earlier stages of disease. When that day comes, the results ought to be still more striking.

(4) Above all else, it should be clearly understood that the object of all hypnotic treatment ought to be the development of the patient's will power, of his own control of his own organism. Many illnesses represent the culminating point in a life which has been characterized by

a lack of discipline and self-control. While attention is given to physical culture, the emotional side is too often neglected; but much disease would be prevented if we could develop and control moral states just as an athlete does physical ones.

Meanwhile, it is not the specialist, like myself, whose practice is confined to unfavourable cases, who is likely to get the best results. It is the general practitioner, who can choose suitable cases from amongst his own patients—patients whose confidence he has already gained.

A society of medical men interested in hypnotism has recently been formed, and already numbers over fifty members. Most of these are general practitioners, and some of them have obtained as good results as I got in Yorkshire some nineteen years ago.

DISCUSSION.

Dr. J. Gray Duncanson said it was true, as Dr. Bramwell had remarked, that hypnotism was too vast a field to be dealt with adequately in one afternoon, but it would be agreed that Dr. Bramwell had gone over it in the time at his disposal in a most thorough and interesting manner. He thought all medical men should approach the subject in a liberal spirit, not forgetting the work and writings of those who had gone before. The nomenclature of the subject was very unsatisfactory. By mesmerism was generally meant what some writers called animal magnetism—the induction of certain hypnotic conditions by means of passes; but Mesmer did not himself use passes at all. Members had also been reminded that many things had to be learnt again and again, owing to this neglect of the past history of hypnotism. The famous French mesmerist, Le Baron du Potet, knew that cures could be effected without sleep being induced, and he, like Esdaile and Elliotson, adopted a form of hypnosis by means of passes. He (Dr. Duncanson) was not sure that the modern school of suggestion was absolutely correct. He did not altogether agree with the older writers that an influence or fluid passed from the operator to the subject, but he thought there was a subtle suggestion or force induced in the subject by means of the passes. Probably no man before or since had produced the absolute insensibility to pain which was accomplished by Esdaile. One of the authorities, and he was a mesmerist, held the theory that: "There is an influence of some kind that passes from one person to another when one of the two is mesmerized." He said: "Never mesmerize people in health." His rule was: "Never mesmerize anyone save for medicinal purposes, and not more than is necessary for the cure of disease." Unlike the martyrs in the cause, Esdaile was very popular. In connection with the production of hypnotism or allied states by means of passes, his brother, Dr. J. C. Duncanson, had done a certain amount in regard to animals. He had a very active bulldog which was not at

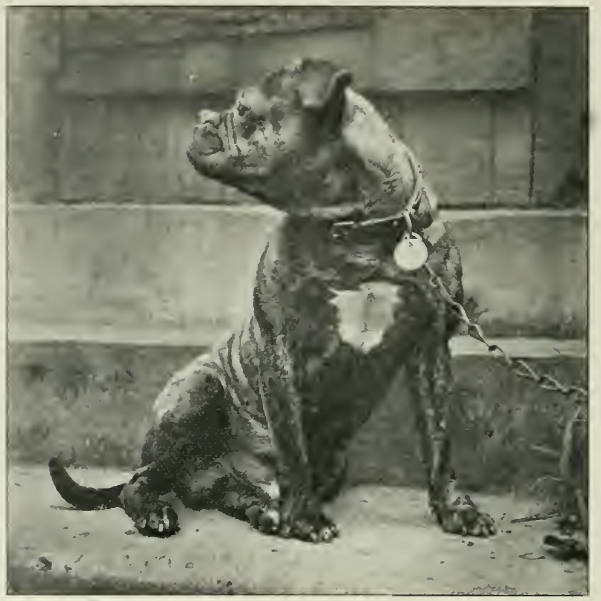

FIG. 1.
Normal condition.

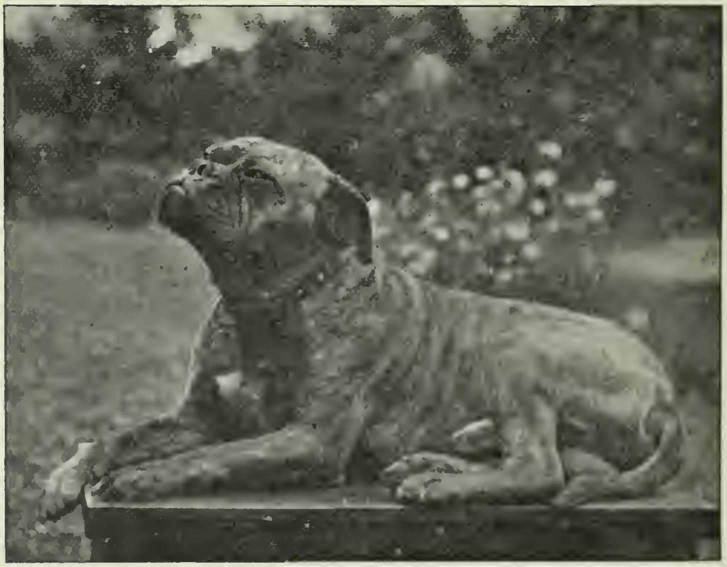

FIG. 2.
Mesmerised condition.
(To illustrate Dr. J. Gray Duncanson's remarks on Hypnotism.)

all morose or dull, and he (the speaker) exhibited two enlargements from photographs (*see* figs. 1 and 2), one in its ordinary state and the other in a mesmeric condition. This was induced by getting the dog to look upwards and inwards with a slight squint. The animal could be seen to be quite flaccid, and the wrinkles and furrows of the face had disappeared. He was allowed to remain so for from four to eight minutes and might have been left in the condition for an indefinite time until roused by blowing upon him and handling him. That seemed to support his view that there was something more than mere suggestion, and he would like to hear a little more on that point. He believed Braid marked the line of demarcation between the old mesmerists and the school of suggestion. Braid did not distinguish between the soul and the mind, and he believed he knew nothing of the subconscious personality; but his work had been of the utmost importance.

Dr. E. B. SHERLOCK asked whether Dr. Bramwell had had any unsatisfactoy results in his treatment by hypnosis. He had followed the matter up, and endeavoured to learn what the hypnotic process amounted to, and in the course of his study he went to Charcot's clinique in Paris, at the Salpêtrière, but was surprised when he was told that the practice of hypnotism had been almost entirely discontinued. Dr. Rose had kindly taken him round the wards and explained the hypnotic side of the treatment, mentioning that the practice had been largely given up because the results were unsatisfactory. He had said that in one of his own cases, for example, the attempt to hypnotize a woman had determined a severe hysterical seizure, and that as hypnosis might do more harm than good it was only employed in very special cases.

Dr. BRAMWELL, in reply, said the question whether there was anything more in hypnotism than suggestion, *i.e.*, some physical influence, such as Esdaile believed in, had been frequently discussed. Some still held this "physical influence" view, but, personally, he believed that suggestion alone was responsible for the results. It was true that Esdaile invariably used passes, but it was a mistake to suppose that since his time no one had been able to induce profound anæsthesia. Many cases of serious operation had been reported in recent years by Continental surgeons who employed hypnotism. It is true that no one had been so universally successful as Esdaile: he, apparently, was able to induce anæsthesia whenever he wished. It was a mistake to suppose that the researches of the Nancy School had carried them to a point far beyond that reached by Braid. Few seemed to be aware that Braid, in one of his later pamphlets, advanced theories in reference to what he described as "alternating consciousness" and "double consciousness," which differ little from the views published by Professor William James, of Harvard, and the late Frederic Myers. Further, until recently, the Nancy School believed in the possibility of suggested crime; Braid held an entirely opposite view, and recent observations show that he was correct. In answer to Dr. Sherlock, Dr. Bramwell said he was not surprised that bad results had occurred at the Salpêtrière. There experiment, not cure, was the main object, and most of the results had been obtained with some half-dozen patients, who were experimented on again and again. The violent methods of inducing hypnosis employed there were likely to cause hysterical outbursts in patients subject to hysterical attacks. It was possible to do harm with any form of treatment if it were used in the wrong way, even with drugs or the knife. A patient ought never to be made the subject of an experiment, no matter how trivial; a patient should be considered a sacred charge. All Dr. Bramwell's experimental hypnotic work had been done on healthy people. Fortunately, the Charcot School of hypnotism was dead, and no one had paid any attention to it for years. At Nancy, the quiet, ordinary, everyday tone of the whole performance formed a marked contrast to the picture drawn by Binet and Féré of the morbid excitement shown at the Salpêtrière. The patients told to go to sleep apparently fell at once into a quiet slumber, then received their dose of suggestions, and, when told to awake, either walked quietly away or sat for a little to chat with their friends, the whole process rarely lasting longer than ten minutes. When I first visited Nancy, two little incidents, illustrating the absence of all fear in connection with hypnotism, interested me greatly. A little girl, aged about 5, dressed shabbily, but evidently in her best, with a crown of paper laurel leaves on her head, and carrying a little book in her hand, toddled into the sanctum, fearlessly interrupted the doctor in the midst of his work by pulling his coat, and said: "You promised me a penny if I got a prize." This, accompanied by kindly words, was smilingly given, incitement to work having been evoked in a pleasing, if not scientific, way. Two little girls, aged about 6 or 7, no doubt brought in the first instance by friends, walked in and sat down on a sofa behind the doctor. He stopped for a moment in his work, made a pass in the direction of one of them, and said: "Sleep, my little kitten," repeated the same for the other, and in an instant they were both asleep. He rapidly gave them their dose of suggestion, and then evidently forgot all about them. In about twenty minutes one awoke and, wishing to go, essayed, by shaking and pulling, to awaken her companion, her amused expression of face, when she failed to do so, being very comic. In about five minutes more the second one awoke and, hand in hand, they trotted laughingly away. In speaking of possible so-called dangers, Dr. Bramwell, while admitting that the sensational experiments at the Salpêtrière were likely to induce hysterical symptoms, drew attention to the fact that Professor Forel, one of the foremost alienists in Europe, who has now abandoned asylum practice to devote himself to hypnotic work, asserted that neither he nor Liébeault, Bernheim, Wetterstrand, van Eeden, de Jong, nor any of the other followers of the Nancy School, had ever seen a single instance in which mental or physical harm, even of the most trivial nature, had been caused by hypnotism. No complete record of their cases had been published, but their number certainly exceeded 50,000.

Therapeutical and Pharmacological Section.

April 28, 1908.

Dr. T. E. Burton Brown, C.I.E., President of the Section, in the Chair.

Remarks on Rheumatism and Chorea in Childhood.

By Leonard G. Guthrie, M.D.

This subject may seem well-worn and even threadbare in this country, and I must confess at the outset that I have nothing new to communicate. I have no fresh theories to bring forward, no original observations to record, and no novelties in the way of treatment to extol. I can only put before you views which I have formed as the result chiefly of personal experience, and hope that the discussion which follows will enlighten myself as well as others.

NATURE OF RHEUMATISM.

I share the common opinion in this country that rheumatism is a specific disease and not merely a "fortuitous concourse" of symptoms. The symptom-complex of rheumatism comprises pains and tenderness in muscles, tendons, joints and nerves, with or without articular or peri-articular or tendinous effusions, certain erythematous skin eruptions, outbreaks of subcutaneous nodules and recurrent sore throat. Acute attacks are associated with pyrexia, malaise and gastro-intestinal disturbance. All these symptoms are in themselves insignificant. It is needless to remark that their grave importance in childhood consists in their frequent association with myo-, peri- and endocarditis, which may speedily end or cripple life. In addition, I regard chorea as essentially a manifestation of rheumatism.

Many speak of rheumatism as a specific *infectious* disorder; but there is no evidence that it has ever occurred in epidemics or that it is directly communicable from one person to another. Yet it is highly probable that rheumatism is due to the presence of some microbic agency, which, however, is harmful only to certain constitutions. In other words, hereditary predisposition to the complaint must be a factor. There is a rheumatic diathesis, just as there are tuberculous and gouty diatheses. The specific nature of the organism concerned in the production of rheumatism is still *sub judice*. Not being a bacteriologist, my opinion in the matter is valueless. I can only say that to my mind the failure of many competent bacteriologists to find the so-called rheumococcus invalidates the conclusions of clinicians who discover it invariably. It seems possible, as Andrewes and Horder have suggested, that common saprophytes, such as *Streptococcus salivarius* and *Streptococcus fæcalis*, may become rheumococci in those who are predisposed to rheumatism.

MINOR MANIFESTATIONS OF RHEUMATISM.

Sore Throat.—Children under 3 years of age rarely make complaint of sore throat, even when on examination the fauces are found covered with diphtheritic membrane. Therefore it is possible that pharyngitis of various kinds may pass undiscovered in young children. I have no doubt, however, that recurrent sore throat is common in rheumatic children. Whether a rheumatic sore throat can be recognized as such is another matter. Some regard any form of tonsillitis, from simple enlargement or follicular inflammation to quinsy, as evidence of rheumatism. Crandall[1] (referred to by Blackader) speaks of "a soft, almost purulent exudate on boggy greyish tonsils" as characteristic. Blackader describes "a diffuse inflammation extending to the pillars of the fauces, sometimes to the posterior wall of the pharynx, and associated with pain on deglutition," as a manifestation of rheumatism. I do not regard any form of tonsillitis as pathognomonic of rheumatism. The subjects of adenoid vegetations and enlarged tonsils are not, I think, especially liable to rheumatism. There is, however, a condition of general relaxation with a bluish red appearance of the faucial mucous membrane, together with slight catarrhal exudation, which seems to be more frequently associated with rheumatism than other forms of sore throat. It may or may not be accompanied by pain on swallowing. The bearing of sore throat in general on rheumatism needs discussion. Some hold that it is evidence of a faucial and tonsillar mode of invasion. Others that it is a local manifestation of rheumatism itself. I myself incline to the view that sore throat of any nature is an indication of lowered health and vitality which favours the action of the rheumatic poison, whatever that may be.

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[1] *Brit. Med. Journ.*, 1906, ii., p. 926.

Cutaneous Manifestations.—Erythema multiforme is, I believe, of definite rheumatic nature. Erythema nodosum I regard as a distinct affection. I have rarely seen it in connection with endocarditis, arthritis, or any rheumatic stigmata. I doubt the existence of a purpura rheumatica, and believe it has been regarded as rheumatic only on account of swelling and tenderness in the neighbourhood of joints, which are really the result of hæmorrhagic effusions.

Subcutaneous nodules are undoubted manifestations of rheumatism. I have made no statistics as to their frequency, and it is a curious fact that, although the supply of cases of articular rheumatism, endo- and pericarditis remains fairly constant throughout the year, the exhibition of nodules is extremely variable. Many months may pass without a single nodule being seen in hospital wards, then a series of cases may occur, all of which develop copious crops of nodules. They make their appearance suddenly from day to day for weeks together, until they number scores or even hundreds. Many quickly melt away, but some persist for months or years. Their favourite sites are in the sheaths of tendons, especially about the wrists, ankles, and knuckles, knees and elbows; and they grow largest in the course of the superior occipital curves, where they may attain the size of cherries, and on the spinous processes of the vertebræ. They are unassociated with pain or tenderness, pyrexia or any constitutional symptoms. Children will often amuse themselves by counting and displaying them as they arrive. When present in considerable numbers they are, in my experience, always in cases of advanced peri- or endocarditis. Although they are doubtless evidence of an active toxæmia, I do not share the view that they are necessarily of grave omen. They may appear in cases which prove rapidly fatal, but I have often seen them come and go in children who recovered sufficiently, at all events, to leave the hospital. The structure of the nodule is interesting. Its morbid anatomy resembles that of the vegetation on cardiac valves. Thrombosis in minute vessels is followed by exudation of lymph, cells and blood, which, if not reabsorbed, leads to formation of nodules of fibrous tissue.

Epistaxis, as Dr. Sidney Phillips has pointed out, is not uncommonly associated with articular effusions. It usually occurs in conjunction with, and perhaps in consequence of, the relaxed and congested state of the fauces already mentioned. But it is not uncommon in cases of mitral regurgitation.

AGE INCIDENCE OF RHEUMATISM.

It is generally, and in my experience rightly, held that articular rheumatism is rare in children aged under 5. Yet, as Dr. Poynton[1] remarks, it is difficult to understand why, if infective agents determine the active disease, the occurrence of infection should be unusual during the first five years of life. No doubt it is true that definite arthritis is extremely rare in children aged under 5, but other symptoms of rheumatism, such as pains and tenderness of joints, muscles and tendons, endocarditis and chorea are far from uncommon below that age.

In Dr. Poynton's list of fifty-two cases of rheumatism in children aged under 5, thirty-five were aged between 4 and 5, but most of these were aged nearer 5 than 4. Ten occurred between the age of 3 and 4; four between the age of 2 and 3; three between the age of 1 and 2. One, aged 10 months, suffered from chorea and endocarditis, but not from arthritis.

In a series of ninety-six arthritic cases of my own, the age incidence was as follows:—

2 to 3 years = 1
3 ,, 4 ,, = 2
4 ,, 5 ,, = 2
5 ,, 6 .. = 7

6 to 7 years = 7
7 ,, 8 ,, = 13
8 ,, 9 ,, = 10
9 ,, 10 ,, = 11

10 to 11 years = 15
11 ,, 12 ,, = 19
12 ,, 13 ,, = 9

Thirty-seven of these were male and fifty-nine female.

Note.—At Paddington Green Children's Hospital, where these cases occurred, boys are admitted up to 12 and girls up to 14. It will be noted that the age incidence shows a steady rise, the greatest number of cases occurring between 11 and 12.

In children aged under 5, were the minor manifestations of rheumatism considered, the disease would no doubt be found to be far more frequent than is generally supposed to be the case. But it is seldom that one meets with more than a vague history of fleeting pains, languor, and so forth in children under 5 who attend the out-patient department. Unless signs of endocarditis or chorea are present, the symptoms rarely render admission to the hospital necessary or justify the diagnosis of rheumatism.

In children aged under 3 acute articular rheumatism should not be diagnosed until scurvy, congenital syphilis, poliomyelitis, spinal caries, osteomyelitis, tuberculous arthritis and septicæmia have been excluded. In children aged under 3, again, cardiac signs and symptoms are far more frequently due to congenital malformation than to rheumatic infection, and choreiform movements are always more suggestive of organic cerebral disease than of true Sydenham's chorea.

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[1] *Quarterly Journ. Med.*, April, 1908, i., No. 3., p. 225.

TREATMENT OF ACUTE RHEUMATISM.

Articular rheumatism is but a trivial and transitory affair in young children; they do not pass through six weeks of agonizing pain, visiting every joint in turn, and many at once, as adults do. In children aged under 12, however severe and widely spread the arthritis may be at first, it usually subsides within a week. After forty-eight hours rest in bed with bandaged joints and a few doses of salicylate of soda, pain is as a rule relieved, effusions disappear, and the temperature falls. Were it not for the imminent danger and frequent occurrence of myo-, endo- and pericarditis, the treatment of articular rheumatism, as well as that of minor manifestations of rheumatism, might be lightly dismissed.

In a large out-patient practice it is impracticable that every child who has recently seemed indisposed, and has complained of pains in its limbs, should be treated as though seriously ill. But whenever there is definite evidence of arthritis or of cardiac affection, however slight, rest in bed should be prescribed for at least three weeks. I cannot claim that rest in bed will prevent cardiac implication, but common sense suggests, and experience shows, that it may be greatly aggravated and perhaps rendered irremediable in children who are allowed to run about and forced to go to school in these conditions.

Use of Salicylates.—Salicylates relieve pain and lower temperature. I do not think they cure rheumatism, and am sure that they do not avert cardiac affection. Indeed, from their depressant effect, I think they are detrimental in rheumatic heart disease. Those who advocate large doses of the drug assert that the nausea, languor, pallor and prostration which commonly occur under its use are due to the disease and not the remedy. But I am convinced that this is not so, and that these symptoms will often disappear on discontinuance of the drug. It is said that the addition of bicarbonate of soda prevents the onset of symptoms of poisoning by salicylate of soda, such symptoms being those of acute acid poisoning, namely, acetonuria, vomiting, thirst, air hunger, delirium, drowsiness, coma, which may end in death. I do not believe that bicarbonate of soda is an antidote to fatty acid intoxication, and I therefore deprecate the risk of ignoring the possibility of poisoning by salicylates in large doses on the ground that an antidote is being given at the same time. I admit that some children seem remarkably tolerant of salicylate of soda. Perhaps in such cases the drug is not absorbed. But admitting the interesting fact that certain children can take enormous doses with impunity, I have failed to convince myself that they are any the better for doing so. Modern improvements in chemistry have rendered synthetic preparations as pure as the natural salicylates. I have not discovered any advantage in using salicin or the much-vaunted acetyl-salicylic acid or aspirin over sodium salicylate.

On several occasions recently I have had to discontinue aspirin on account of the nausea and vomiting which accompanied its use. The fact that it may produce hæmaturia, to my mind, is sufficient to condemn its administration in large doses. Salicylate of quinine is not so efficacious in reducing pain in acute rheumatism as salicylate of soda, but it has the advantage of combining a tonic with an anodyne.

TREATMENT OF RHEUMATIC CARDITIS.

If one sees the pathological condition of cardiac inflammation after death one recollects without surprise that treatment was useless during life. It may be possible to stimulate, regulate or calm the action of the heart by drugs, but cardiac tonics and sedatives will not prevent formation of pericardial adhesions, inflammation of the valves, and their subsequent incompetency due to cicatrization.

When the myocardium is itself inflamed, drugs such as digitalis seem powerless for good. The test of the efficacy of digitalis is gauged by its effect on the pulse. If the pulse remains persistently rapid under its use, digitalis is doing no good; and if the pulse becomes small, hard, and irregular, it is doing harm. By increasing peripheral resistance in the arterioles without stimulating and slowing the action of the heart itself, digitalis may give rise to acute distension of the right heart, with dyspnœa, lividity, pulsation of the cervical veins, engorgement and enlargement of the liver, followed rapidly by ascites and general dropsy. However, acute distension of the right heart is often the result of backward pressure from mitral regurgitation, apart from the use of digitalis, and in such cases the drug, combined with squill and mercury, is invaluable. Its administration should be preceded by dry-cupping, leeching, or venesection, and these remedies alone are often strikingly successful in cases of acute dilatation of the right heart. Dry-cupping to the chest is, again, useful in relieving the pulmonary congestion which so often is the effect of a labouring and incompetent heart.

I have occasionally seen good results in cardiac ascites and dropsy from the use of theocin-sodium-acetate, combined with digitalis. But unfortunately it often excites serious vomiting. I do not think it is in any way superior to Baly's pill or equivalent powder in such cases.

Acute pericarditis sometimes seems to subside after application of flying blisters to the præcordia. But disappearance of friction and relief of pain may be due to effusion of fluid, which prevents inflamed structures from rubbing together. Severe pain, especially if accompanied by restlessness and vomiting, is a sign of gravest omen. Morphia is the only remedy which is of service. Indeed I am in the habit of keeping all cases of heart disease in children, when pain, distress, restlessness, and insomnia are present, under the influence of opium, for I know not what else to do for them.

CHOREA.

The close relationship of chorea to rheumatism is now generally recognized. This relationship was long disputed on the ground that most severe and even fatal cases of articular and cardiac rheumatism may run their course without any signs of chorea, whilst the most intractable and violent forms of chorea may occur in those who show no other signs of rheumatism. Chorea, again, which followed immediately on fright or shock or traumatism was held to be in a different category from that which occurred in subjects undoubtedly rheumatic. But Dr. F. E. Batten, who followed up the history of 115 cases of chorea treated at Great Ormond Street, found that at least 20 per cent. of those who at the time showed no signs of rheumatism developed it within six years. In many cases of apparently non-rheumatic chorea there is a strongly marked family history of rheumatism, and in many, previous minor manifestations of rheumatism have been unrecognized as such.

The difficulty in associating "fright" cases of chorea with a rheumatic origin is in part occasioned by the confusion which has reigned between tics, or so-called "habit spasms," and true Sydenham's chorea. The movements in tic and chorea are, however, absolutely unlike each other. It may be conceded, nevertheless, that tics and true chorea are sometimes met together. Tics, or habit spasms, are undoubtedly the consequence of emotional distress. Personally, I see no difficulty in assuming that "fright" may give rise to rheumatism or chorea, for fright, emotional disturbance of any kind, traumatism, exposure to cold and wet will all alike lower and disturb our vital and metabolic processes.

Given a predisposition to rheumatism and the presence of the rheumatic germ or poison, a shock of any sort may determine an attack of rheumatism or chorea.

In cases of chorea attributed to fright or shock, traumatism or chill, it is often hard to decide which of these agents played the major part. For instance, a boy developed chorea three days after playing truant from school and getting wet through. Was the chorea due to chill, to fear of traumatism, or to traumatism which he actually sustained at the hands of his parents on arriving home? Again, a little girl after being put to bed was found on a cold winter's night cowering on the landing in alarm at the noise made by a brawling drunken woman on the floor below. Next day she became choreic. Was the chorea due to fright, exposure to cold, or both?

The same question applies to another girl, who fell into the water up to her neck, and was much frightened in consequence. Chorea followed a few days later. A week previous to her immersion she showed no signs of chorea. In the histories of children admitted to hospital for chorea I find instances such as follow: "Was kicked in the groin a week ago"; "Came home from school crying, with a stiff neck"; said "Another boy had jumped on his back and hurt him"; "Was hit on the head at school."

Clement Lucas[1] has recorded the case of a child, aged 11, who developed general chorea half an hour after being run over by a cart and sustaining fracture of the right humerus. An apparently healthy girl, aged 10 (whose case has been published by G. A. Sutherland), underwent an operation for radical cure of hernia. She became extremely collapsed whilst under chloroform. A few days later she had an attack of acute tonsillitis, succeeded by most severe endo- and pericarditis and equally violent chorea. I have also known a boy who became collapsed under chloroform whilst undergoing an operation for strabismus, and a week afterwards he developed acute arthritic rheumatism. Such cases seem to support the view that shock, whether emotional or traumatic, may light up rheumatism in those who are susceptible and in whom the rheumatic poison is present.

In the causation of chorea, however, I believe that the paramount factors are an emotional temperament and nervous instability. These factors are present in all cases of chorea, and their absence accounts for cases of undoubted rheumatism which are yet unaccompanied by chorea.

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[1] *Trans. Soc. Study of Dis. in Child.*, 1901-2, ii., p. 74.

Chorea is, in short, a psychomotor manifestation of rheumatism occurring only in rheumatic children who are psychasthenic or mentally unstable. The movements are not those which result from mechanical irritation of the motor cortex. They are not in the least like the convulsions seen in Jacksonian epilepsy.

The movements in chorea imply disturbance of the highest cerebral level. Uncontrollable impulses prompt the exaggerated and unnecessary performance of all the normal movements of which the body is capable; and these movements are at one and the same time thwarted by equally uncontrollable impulses to perform movements of opposite character. The facial movements are not spasms. They represent expressions of all the mental emotions and suggest attempts to express each and all of them at once in spite of the absence of these emotions. They are therefore evidence of psychical disturbance, and I believe that recognition of this fact has an important bearing on the treatment of chorea. The expression "uncontrollable impulses" implies, no doubt, "defective inhibition," and it may be said with certainty that in the process of recovery either the impulses to perform movements become less peremptory or the power to inhibit them is restored.

ANALYSIS OF 114 CASES OF CHOREA.

For comparison with the statistics of others I have analysed 114 cases of chorea in respect to age, sex, family and personal history of rheumatism, seasonal incidence and duration.

Age.—Up to the age of 8 to 9, boys are rather more frequently affected than girls; but beyond this age there is a marked preponderance of female over male subjects of chorea. The actual figures are as follow:—

	Males	Females		Males	Females
Between 2 and 3 years	1	0	Between 8 and 9 years	10	5
,, 3 ,, 4 ,,	0	2	,, 9 ,, 10 ,,	7	17
,, 4 ,, 5 ,,	0	2	,, 10 ,, 11 ,,	1	17
,, 5 ,, 6 ,,	5	1	,, 11 ,, 12 ,,	5	16
,, 6 ,, 7 ,,	3	5	,, 12 ,, 13 ,,	0	4
,, 7 ,, 8 ,,	4	7	,, 13 ,, 14 ,,	0	2

Thirty-six were male and seventy-eight female.

In another series of fifty cases of chorea I find that sixteen were male and thirty-four female, thus giving again a proportion of two girls to one boy. The proportion estimated by other observers is three girls to one boy, but at this particular hospital boys aged over 12 are not admitted, whilst girls are admitted up to the age of 14. The age incidence shows that chorea is rare under 5 and extremely so under 3.

Family history of rheumatism was marked in fifty-four cases (47·3 per cent.); absent in forty-eight cases (42·1 per cent.); doubtful in twelve cases (9 per cent.).

Personal history of rheumatism was marked in seventy-two cases (63·1 per cent.); absent in thirty-seven cases (32·4 per cent.); doubtful in five cases (4 per cent.).

Cardiac affection in 114 cases of chorea was marked in fifty-nine cases (44 per cent.); absent in forty-nine cases (42 per cent.); doubtful in six cases (4 per cent.).

Seasonal incidence of chorea: During January, February, and March twenty-one cases were admitted; during April, May, and June twenty-eight cases were admitted; during July, August, and September twenty-five cases were admitted; October, November, and December forty cases were admitted.

Thus chorea seems to be more common during the last than in any other quarter of the year; whereas it is usually considered, in London at all events, to be most common during July and August. The discrepancy is probably due partly to the fact that the hospital is partly closed for cleaning in August. Possibly rheumatism is excited in the summer months and chorea ensues in the winter.

Average Duration.—The average duration of stay in hospital is five to six weeks. In a few it has been as many months, but these have been either severe relapsing cases or cases complicated by grave heart disease. The length of stay in hospital gives, of course, only a rough estimate of the duration of the illness. If dated from the onset the average duration appears to be about six to seven weeks.

Relapsing Cases.—In 114 cases seventy-six were admitted in a first attack, twenty-three were admitted in a second attack, and thirteen were admitted in a third attack. In two cases there was a doubtful history of previous attacks.

As regards the ages of patients admitted for first, second, and third attacks:—

ADMITTED FOR FIRST ATTACK.

Between 2 and 3 years ...	1	Between 8 and 9 years ...	7
,, 3 ,, 4 ,, ...	1	,, 9 ,, 10 ,, ...	14
,, 4 ,, 5 ,, ...	2	,, 10 ,, 11 ,, ...	13
,, 5 ,, 6 ,, ...	6	,, 11 ,, 12 ,, ...	13
,, 6 ,, 7 ,, ...	6	,, 12 ,, 13 ,, ...	2
,, 7 ,, 8 ,, ...	11		

ADMITTED FOR SECOND ATTACK.

Between 6 and 7 years	1	Between 10 and 11 years	2
,, 7 ,, 8 ,,	1	,, 11 ,, 12 ,,	6
,, 8 ,, 9 ,,	4	,, 12 ,, 13 ,,	4
,, 9 ,, 10 ,,	5		

ADMITTED FOR THIRD ATTACK.

Between 8 and 9 years	3	Between 11 and 12 years	2
,, 9 ,, 10 ,,	4	,, 12 ,, 13 ,,	1
,, 10 ,, 11 ,,	3	Doubtful	2

Thus first attacks were most frequent between the ages of 7 and 12 but occurred at any age from 3 upwards; second attacks were not met with before the age of 6, while third attacks were not seen below the age of 8.

The number of cases investigated is small, and I have not attempted to analyse a longer series, but the results bear out the general impressions which I had formed before undertaking this inquiry.

TREATMENT OF CHOREA.

My conception of Sydenham's chorea is that it is the resultant action of an unknown rheumatic poison on the unstable and excitable higher centres of a neurotic individual's brain. The rheumatic nature of chorea is shown in the frequency with which it is associated with a family and personal history of rheumatism and with the cardiac affections which are generally attributed to rheumatism.

In chorea the neurotic or emotional element is obvious, and the severity of the complaint depends more on it than on the degree of rheumatic infection present. Hence the most violent forms of chorea are often associated with the least evidence of rheumatism.

If, as I have contended, we have no drugs which cure rheumatism, it follows that none will cure chorea, which is a functional neurosis or protracted "nerve storm" induced by rheumatic toxæmia, but often long outlasting its cause. The treatment, therefore, of chorea must be directed chiefly to the psychical or emotional condition present.

The "nerve storm" itself, characterized by violent persistent and uncontrollable movements, tends to wear itself out spontaneously in the course of one to three weeks. In exceptional cases it may last a week or so longer. At the end of this time, in severe cases, the spontaneous movements may practically cease, but the paretic form of chorea may take their place. Attempts at voluntary actions are only represented by slight shrugs and twists, or are rendered futile by absence of precision and wild ataxy. There is often a condition of great weakness and prostration. Mutism and a fatuous mental condition are common; night terrors, screaming attacks, and even mania may occur at this stage. More commonly, however, at the end of one to three weeks the general movements may still continue, but on testing it is found that they are controllable by effort of will. Sometimes they are increased or only present when notice is taken of the patient; sometimes they only occur when the patient is left unwatched. Sometimes the child will be perfectly still, but all voluntary movements are rendered impossible by ataxy. It is important to recognize these various conditions of "residual chorea." If untreated they may persist indefinitely. As soon as it is ascertained by testing that some power of voluntary control of choreic movements is present, the treatment consists in encouraging the patient to exert it. The suggestion methods, as used by hypnotists, should be adopted. Ataxy on voluntary movement should be treated by modified Frenkel's methods, as used in tabetic incoördination; building wooden blocks, setting out tin soldiers and the inhabitants of the Noah's Ark, are useful exercises, and most of the apparatus used in kindergartens may be employed in order to assist in regaining precision of movements. Children at this stage should not be kept in bed too long. I have often noticed that they cease to improve if they fret at not being allowed to get up. Care should be exercised, however, not to overtax their energies on first getting up. Over-exertion and excitement will often bring about a relapse. As soon as they are able to stand steadily and walk without assistance a course of simple drills and exercises will soon complete recovery. A slight continuous pyrexia, which is often regarded as a reason for confinement to bed, will often subside if the patients are permitted to be up and dressed.

TREATMENT OF ACUTE (OR STHENIC) STAGE OF CHOREA.

Rest in bed, isolation, quiet, careful nursing and feeding, protection from injury by padded bed-sides, cotton wool bandages, and sometimes by splints, are requisite in the treatment of all acute and severe cases. When there is much emotional excitement, and sleep is prevented by violence of the movements, hypnotics and various nerve sedatives are necessary. Bromide and chloral are the drugs I prefer. A dose at night only is usually sufficient, but sometimes it must be repeated twice or three times daily. I have used monobromide of camphor, in 1 gr. to

4 gr. doses, with fairly good effect. Extract. cannabis indicæ, $\frac{1}{8}$ gr. to $\frac{1}{2}$ gr., with ext. physostigmatis, $\frac{1}{26}$ gr. to $\frac{1}{6}$ gr., has sometimes appeared beneficial. Hyoscin hydrobromate is a dangerous drug to use, but in maniacal cases may be of service in $\frac{1}{100}$ gr. doses *sub cutem.* Antipyrine as a nerve sedative has been strongly recommended, but I have not been particularly struck by its efficacy. Chloretone has been strongly recommended by Dr. Essex Wynter. Whenever depressants are used, stimulants—alcoholic and otherwise—are necessary. Fortunately, chorea never occurs in conjunction with articular inflammation, otherwise the patient's sufferings would be indeed pitiable. But pains in various parts are frequent in chorea, and they are relieved by sodium salicylate or aspirin, as in the case of acute rheumatism.

Twenty years ago, when treatment by arsenic was much in vogue, I had frequent opportunities of observing its results, whether given in large doses or by the "intensive" method. I could never satisfy myself that it did any good, and I often saw it do harm by causing acute gastritis, and, not rarely, by producing arsenical neuritis. It is true that some children seemed remarkably tolerant of the drug, but the average duration of chorea was not materially altered by its use. On comparing a series of forty-four cases of chorea treated by arsenic with one of sixty-two cases treated without arsenic, the average duration of stay in hospital was, in the former, five weeks, in the latter 5·3 weeks. Arsenic, however, in small doses is a valuable nerve tonic, and I constantly use it to aid convalescence.

In the treatment of paretic cases (chorea mollis), nutritious food, sometimes administered by nasal tube, must be supplied. Alcoholic stimulants in abundance may be required. The patients are usually emaciated and much reduced in strength. The best results are attained by massage and passive movement in such cases.

Aspirin as a specific for chorea has been strongly recommended by Drs. Cecil Bligh Wall and James Burnet in the *Transactions of the Therapeutical Society* for 1905 and 1907.

Dr. Cecil Bligh Wall found that of fifty cases treated by aceto-salicylic acid all got well in less than three months; forty-nine (98 per cent.) in less than two months; thirty-seven (74 per cent.) were pronounced cured in one month or less. He does not mention the existence of cardiac or arthritic complication in these cases. I must confess that I am not convinced by these figures that aspirin is a cure for chorea.

I should not regard any case not cured in less than three months as a testimonial to the methods of treatment adopted. The average duration of stay in hospital of choreic cases in my own experience is about five weeks. This average would be much lowered were it not for those patients who were detained for three or even six months on account of relapses or severe heart disease.

Mortality in Chorea.

The mortality of chorea in childhood is usually stated to be 2 per cent. I have never known death to occur from uncomplicated chorea, however violent and exhausting the movements may have been. The cause of death has always been either endo- or pericarditis or septicæmia. One fatal case was that of a girl, aged 7, who had no personal or family history of rheumatism or endocarditis. A month after admission the choreic movements had almost disappeared, but severe oral sepsis took place. A rash of mixed scarlatinal, morbilliform, and urticarial appearance covered the whole body, coming and going and varying in intensity for twelve days. The temperature varied between 103° F. and 105° F. The rash was followed by profuse desquamation, and the patient died after a fortnight's illness from septicæmia, probably of streptococcal type.

Dr. Herbert French, in an interesting communication on "Chorea Gravidarum,"[1] has shown that the mortality from the affection is practically confined to pyrexial or septicæmic cases, and this conclusion seems also to apply to juvenile chorea.

Conclusions.

(1) Rheumatism is a specific infective disease.

(2) The factors concerned in its production are: (*a*) inherited predisposition; (*b*) presence of an undetermined microbic or toxic agent which may possibly be innocuous in non-rheumatic subjects (*e.g.*, *Streptococcus salivarius* or *Streptococcus fæcalis*); (*c*) a condition of lowered vitality in the subject, which may be induced by sudden shock of any kind or by prolonged exposure to cold or damp.

(3) Chorea is a rheumatic manifestation which occurs only in those who are also neurotic or emotional. Chorea is a psychical disorder; its exciting causes may be sudden, short, or prolonged emotional disturbance, in addition to the factors concerned in the production of rheumatism itself.

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[1] *Practitioner*, 1906, lxxvii., p. 178.

(4) There is an acute stage of chorea, characterized by uncontrollable impulses to perform violent and contradictory movements. The duration of this stage seldom exceeds one to two weeks.

(5) The acute stage is succeeded sometimes by a condition of profound mental and physical prostration, with cessation of choreic movements (paretic chorea or chorea mollis).

(6) A condition in which the movements continue, but can be restrained and guided by act of will (residual or habitual chorea).

(7) Drugs may palliate, but do not cure either rheumatism or chorea.

(8) The acute stage of chorea needs treatment by rest, quiet, and sedatives. Salicylates in moderate doses are useful when pain is present. The paretic stage should be treated on ordinary principles, such as are used during convalescence from any exhausting illness. The residual or habitual stage of chorea needs treatment by moral persuasion, drill, and exercises.

(9) The practice of poisoning choreic patients by arsenic, salicylates or any other drug is deprecated, but it is admitted that the general prostration induced thereby may appear to shorten the acute stage of chorea.

(10) Chorea is never fatal in itself.

(11) In conclusion, the common and disastrous effects of rheumatism in crippling and shortening life, the futility of all treatment designed to arrest or modify its fell influence, render urgent the need to seek a remedy. Further bacteriological research affords the only ray of hope. Common and non-pathogenic organisms may be pathogenic in certain individuals, and should this be the case we must put our trust in the use of appropriate vaccines.

DISCUSSION.

Dr. CECIL WALL said that he had listened with very great interest to Dr. Guthrie's paper, and he might say that with almost everything said therein he felt himself completely in accord. He knew Dr. Guthrie's views from his previous writings on the subject, and had always looked on him as one of the authorities in the treatment of chorea. When he (the speaker) read the paper that he did about two years ago before the Therapeutical Society[1] on the treatment of chorea by aspirin, it was with full knowledge of Dr. Guthrie's work on the subject, and the experiments in treatment were only undertaken because he felt that Dr. Guthrie's position was a little unsatisfactory. He felt, at all events at the hospital in which he was working (the London Hospital), that they were not getting results as satisfactory as they might have been. The tables he (the speaker) then quoted were tables taken from the records of the London Hospital, and included all cases of chorea of all grades of severity. He might add that usually only the severe cases were admitted to the hospital, but all cases so admitted were taken, whatever the complications present, and the whole number was included in the tables. He relied rather on the number of cases under consideration to exclude error than on any attempt at selection. The majority of cases admitted to the London Hospital undoubtedly got well within eight weeks. Of in-patients treated by arsenic he found that very many were cured within eight weeks, *i.e.*, were discharged from the hospital. That was the way in which he decided whether the patient was cured or not. As to the treatment by arsenic, the number of cases cured in less than two months was 38 per cent. It was a very large number considering the type of cases admitted. Similar figures held for other methods of treatment under discussion—the treatment with salicylate of soda and so forth. It was only when they came to the treatment by aceto-salicylic acid that they found a very marked difference. Certainly there were not quite so many cases, but he found that 92 per cent. of the cases treated by aceto-salicylic acid were discharged from the hospital in less than two months; many were discharged in four weeks from admission, which seemed to him very good. One explanation of the reason that the cases seemed to stay in rather longer at the London Hospital than at Paddington Green was that they took in all cases, whatever their age; he thought it was a well-recognized fact that chorea was a disease that had a much shorter natural course in the younger patients than in older ones. The patients of 15, 16 or 17 admitted to the hospital often had very prolonged attacks. His feeling with regard to the treatment of chorea by aspirin, based as it was on figures which certainly seemed conclusive to him, was confirmed by two more years experience of the treatment, and he still felt very strongly in favour of that treatment, and had persuaded most of his colleagues on the staff of the London Hospital to that view, even though they were of Dr. Guthrie's opinion originally, that no drug had any marked influence on the course of an attack of chorea. He might add, with regard to the dangers of the use of aspirin—the vomiting which Dr. Guthrie alluded to—that he found it was quite possible to avoid it altogether if aspirin were only given when there was food in the stomach. It was quite useless giving aspirin on an empty stomach. It was almost certain to be vomited. The salicylic acid would be given off, the stomach would be irritated and vomiting would result. Given on a full stomach that result practically never happened. There was another point, that aspirin should never be given in tablet form, it always should be given in powder. If given in tablets one or two minor accidents might happen. Vomiting sometimes occurred, and in a few cases which he had seen he had discovered traces of blood in the stools afterwards. Sometimes, of course, the tablet passed through unchanged, but blood in the stools he supposed was due to the mechanical irritation of the aspirin crystals. Since insisting on its being given in powder form he had never had any trouble on that score. He had seen, as he thought Dr. Bryant first mentioned, hæmaturia as the result of giving aspirin. In two such cases aspirin was given in inordinately large doses by an over-keen house physician without his knowledge, and certainly without his sanction. One of them, a policeman, came in with acute rheumatism, and for some unknown reason the house physician prescribed 30 gr. of aspirin every two hours for six doses. The hæmaturia lasted about forty-eight hours and cleared up: it did not seem to do the patient any harm at all. Beyond that he had never seen any ill result from aspirin. He did not believe in heroic doses, because he thought with moderate doses he could get fairly good results. With regard to the other drugs that Dr. Guthrie mentioned, in violent cases he thought it was wise to use sedatives. Chloral and chloralamide were the two which he preferred. He thought the use of bromides was almost as bad as, if not worse than, the use of arsenic in the treatment of chorea; not that small doses of bromides would do any harm, but there was a tendency, when bromides were given, to push them. If there was one drug which it would do more harm to push than another it was bromide. As medical registrar of the London Hospital for two years he learned that. He saw that when bromides were given innumerable dangers and troubles were likely to arise. In certain cases the result of giving bromides had been to produce mania. Any depressing drug like bromide was likely to exaggerate rather than ameliorate the condition. He found that chloral and chloralamide, perhaps combined with a little alcohol, were quite sufficient to secure sleep, even in the most violent cases of chorea. Bromides he always avoided as harmful. Bromides and chloral combined did less good than chloral alone. That, of course, was not merely his own view with regard to the use of chloral. It had been pointed out many times before. There was a theoretical point on which he should like to touch in regard to the paper, and to gain a little elucidation from Dr. Guthrie. He spoke of chorea as a psychomotor disturbance. By that he gathered that Dr. Guthrie rather looked upon chorea as the result of some irritation or some increased activity of a cerebral process. His own view of chorea was different. He followed a view which he believed Dr. Warner originally put forward, that chorea was a condition of reversion to the infantile type; the new-born infant had normally the spontaneous irregular movement of limbs such as was met with in cases of chorea, and in the earlier years of life the cortex gradually gained control over those movements and voluntary coördinated movement was the result; inhibition of spontaneous movement was the power which was conferred upon the cortex during the earlier years of education. If during those years of education some influence arose which upset the equilibrium of the cortex and diminished its inhibitory power, then the lower centres of the nervous system would act spontaneously. He looked on chorea as a condition brought about in that way: that really it was due to a paralysis of the cortical centres allowing the lower parts of the nervous system to act spontaneously and resulting in the reversion in the type of movement to the condition found in the new-born infant. He believed that was the only theory which could adequately explain the many peculiarities of chorea. The age incidence, for one thing, which Dr. Guthrie did not speak of, was one of the most startling peculiarities of chorea. They practically never met with true chorea after the age of 25, apart from some few instances in association with pregnancy. This theory explained very fairly the age incidence of chorea. During all that period of life up to the age of 25 the cortex was gradually being developed. It was not until the age of 25 that the cortex was really firmly established in its functions, and during all that period they could easily imagine that it was in a condition of somewhat unstable equilibrium. If any debilitating influence acted on the developing cortex, whether a toxin or a germ, whether it was a mental overstrain or what not, it was easy to understand that the controlling influence might be diminished and uncontrolled spontaneous movements might result. The theory also offered an explanation of the loss of emotional control in chorea. It was a little different from what, he took it, Dr. Guthrie put forward as his explanation, though he should like to know from him whether he really looked on chorea as due to irritation or due to partial paralysis of the higher centres in the brain.

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[1] *Therap. Soc. Trans.*, 1907, 5th Session, p. 50.

Dr. LEES said that the subject of Dr. Guthrie's paper was one of the most important in the domain of children's diseases. It was a subject, or group of subjects, in which he had taken very great interest during the whole time he was physician to the Hospital for Sick Children—for fifteen years. He felt that the greater part of the statements which Dr. Guthrie had made was entirely confirmed by his own experience. Dr. Guthrie had put before them in a most interesting way many important and valuable facts which were not sufficiently recognized by the profession at large. Of course every man's experience was more or less special, and there were some points in which he did not altogether concur in the conclusions which Dr. Guthrie expressed, but it was a pleasure to recognize that their experience in the great mass of cases was very similar. He agreed with what Dr. Guthrie said about the prognosis of nodules. At first there was a tendency to make a little too much of the evil prognosis of nodules, and a severe eruption of nodules was even regarded as "equivalent to a sentence of death." Now that first view was not unnatural, but it certainly was a little exaggerated. Still he thought—and he gathered that Dr. Guthrie thought—that the presence of many large nodules was an additional grave element in the prognosis. But there were many children with nodules who were very slightly affected by them, and he would add that the prognosis was distinctly better if they were efficiently treated. He wished also to express his concurrence with Dr. Guthrie about the frequent uselessness of digitalis. In the acutely dilated hearts of rheumatic children digitalis did no good; he suspected that it might do harm. Also he would point out the value of Dr. Guthrie's suggestion about bleeding when the right heart was over-distended. The relief given by one or two leeches in such a case was very great. With regard to chorea, he agreed with what the lecturer said about the probability that the occurrence of chorea after fright or injury was really a development of an already present rheumatic poisoning. One remembered somewhat similar occurrences. For instance there was the story of the boy who was hit on the head with a ruler by his teacher at school, and who at once became ill and died of tuberculous meningitis. He also instanced the case of a boy who had received a blow upon his head which was rapidly followed by delirium; meningitis was suspected, but the case proved to be simply pneumonia. In both these cases the specific organism was already present, but the injury was the determining cause which allowed it to manifest itself. With regard to the question of the nervous factor in rheumatism and chorea, he thought that it was not simply that chorea developed in neurotic children, but that the neurotic element in chorea was probably largely the effect of the rheumatic toxin on the brain; and in confirmation of that he pointed out that in many minor cases of rheumatism in children careful examination would detect either evidence of slight chorea or sometimes of the peculiar neurotic tendency to cry without cause which was often so marked in chorea. There might be no obvious choreic movement, but the child had a tendency to burst into sudden crying, which passed off in a few minutes, and he could not tell why he cried. Then there were other evidences of the affection of the higher parts of the brain in chorea, he thought, in addition to those which Dr. Guthrie mentioned, such as the alterations in temper and disposition which were often manifested in the early stage of chorea. Also the speechlessness of a severe chorea was more than simple weakness of muscular movement—it was a definite aphasia. Lastly, hallucinations of vision were sometimes quite distinct in choreic children. All these facts pointed to an action of something or other on the higher centres of the brain. Now with regard to the question of treatment. In the first place he was extremely interested to hear what Dr. Wall told them about aspirin, and asked for information as to the dose employed. He would also like to ask in the same connection the dose of salicylate which Dr. Guthrie was in the habit of using in rheumatism and chorea, because it seemed to him that the question was very largely one of dose; and he must confess that his conclusions as to treatment did not agree with those of Dr. Guthrie. He had for some years been gradually increasing the dose of salicylate given to rheumatic and choreic children, and he was quite convinced—of course he could not expect by this mere statement to convince Dr. Guthrie—but he was personally convinced, and he thought others who had watched the cases were also convinced, that children with rheumatism and chorea recovered much more rapidly and much more completely with large doses of salicylate than they did with a small dose, that there was a very much less frequency of relapse, and that the irregular temperature of 99° F. to 100° F., which went on, sometimes for weeks, in children who were treated by inefficient doses of salicylate, was conspicuous by its absence. He was now convinced that salicylate did much more than merely relieve rheumatic pain. He thought that the day would come when it would be recognized generally that salicylate, either by itself or, as Dr. Wall gave it, in the form of aspirin (which was a salicylate), was a definite specific against the rheumatic process (toxin or microbe—he believed microbe), as distinctly as quinine was a specific against malaria or mercury against syphilis. But this doctrine would not be accepted until practitioners acquired the habit of giving larger doses. Now why did medical men give, as a rule, what seemed to him ineffective doses of salicylate? The reason was this: that there were certain well-known unpleasant symptoms which might be caused by salicylate. When those symptoms occurred the usual practice was to drop the drug, to say it did not suit this patient, and they must try something else. Now he would venture to suggest to them that they should adopt a new method, and instead of giving up the drug they should omit it only for two or three doses, and then recommence with half or two-thirds of the dose to which they had already attained. He believed very strongly that salicylate was much more easily tolerated if a double dose of bicarbonate of soda was given with it. What he found was, that if it were given in that combination at frequent intervals (ten doses every twenty-four hours), until some unpleasant symptom was produced, then omitted, and recommenced at half or two-thirds the dose, it was usually possible with most patients, whether adults or children, to educate the patient until he or she could take without difficulty two or three, perhaps four, times the amount of the dose which at first caused symptoms of poisoning. If they tested that, he thought they would come to the conclusion that the effects of these large doses of salicylate were so distinct that there could be no doubt remaining that salicylate was definitely a specific against rheumatism. If it were a specific against rheumatism, and if chorea was rheumatic, it ought to be a specific against chorea. He said to himself some years ago: "Why is it that the salicylates, which certainly seem to improve rheumatism, do not seem to improve chorea?" and he remembered that if they tried to treat a gumma of the brain by 4 gr. or 5 gr. of iodide of potassium they would have no result. They must give large doses in such cases, and he thought it probable that the same might hold with chorea in children. He had cautiously and gradually increased the doses until he gave these children 300 gr. or 400 gr. of salicylate of soda in the twenty-four hours, and under these large doses of salicylate, with double doses of bicarbonate of soda, he had not found that they were depressed. On the contrary they became more lively and brighter. In rare cases there might be a little depression, but the great majority of cases became brighter and happier while they were taking these large doses. The only really troublesome symptom was vomiting, and that could usually be got over by adopting the method which he had suggested. He thought it would some day be recognized that rheumatism was a definite microbic process with toxic results on the heart and on the brain, and that this toxic process could be definitely arrested by sufficient doses of salicylate of soda administered in the way he had suggested. But it was necessary to add that whether the dose of salicylate given to a rheumatic child was small or large, two precautions were always necessary: the urine must be rendered alkaline and constipation must be prevented. If these two conditions were secured, the dose of salicylate might be rapidly increased; if they were neglected, even small doses might be poisonous. He concluded by drawing attention to the extraordinary value of the icebag in rheumatic pericarditis, and he thought it was very useful also in rheumatic myocarditis. Of course the patient's feet and lower limbs must be kept warm by hot water bottles while the icebag was applied over the heart.

Dr. GORDON SHARP thought the paper was a very singular one, in so far that it was what one might call free from fads, which was a very important point in dealing with such important diseases as chorea and rheumatism. However, he was rather astonished to find that Dr. Guthrie's experience taught him that the salicylates were depressing. He must say, in a fairly long experience, both in hospital out-patient practice and in private practice, he had never found that depression resulted from the use of salicylates, and his experience confirmed that of Dr. Lees that large doses did great good. They gave comfort, they eased the aching joints, and if they gave sleep and comfort to a patient he believed it was the starting-point in cure or recovery, or whatever they might like to term it. As they knew, patients did not like liquids, and he always made sure that his rheumatic patients took liquids. For a child he gave 2 oz. or 3 oz., and for an adult the dose would be 5 oz. in aerated soda-water. With the help of liquids he could get better results with smaller doses, and they undoubtedly did a great deal of good. It was advisable to take care that the patient was rubbed down. As to the use of digitalis it was a curious thing how little they had progressed since the days of Withering. He found that Withering, in his experiences at the end of his book, said: "If digitalis is given when the pulse is cordy it has very little effect, but if the pulse is soft," as he put it, or of low tension as we would say, "then digitalis always did good." Great minds thought alike, and Dr. Leonard Guthrie's experience seemed to be the experience of their great master in the use of digitalis—Withering. He was much astonished that the London experience was that arsenic did no good, but in times gone by he had recommended Fowler's solution after a meal, though the doctor to whom he recommended this was loth to adopt this remedy. His experience, having treated chorea in this way in a large out-patient department, was that the patients did remarkably well. There must be something peculiar in the London constitution that could not stand that very important drug. Of course there were cases that did not recover with arsenic, in the same way as there were cases of acute rheumatism that did not recover under any drug they liked to specify. He was much struck and pleased to hear that Dr. Guthrie had found that there was an intimate connection between rheumatism and chorea. He knew that many authorities thought they were quite distinct affections, but he (the speaker) did not believe it.

Dr. J. GRAY DUNCANSON emphasized the importance of always giving a physiologically pure salicylate of soda, in large doses, at short intervals every two hours and well diluted, and he (the speaker) found in his experience that when administered in combination with bicarbonate of soda and liquor bismuthi, even the most irritable stomach could be brought to tolerate it. He still held to the opinion he expressed in the discussion which followed Dr. Cecil Wall's excellent address on chorea, delivered two years ago before the Therapeutical Society, that in Scotland and the North of England children with chorea, when treated by arsenic, almost invariably recovered quickly; when he was house physician they seldom had cases of chorea in hospital more than three weeks. The cases were at once put on moderate doses of Fowler's solution, which were rapidly increased until physiological effects appeared; and he believed the whole explanation was simply that of environment and racial distinction. The temperament here in London was entirely different from what it was in the North, and he personally had almost given up giving arsenic in chorea, in fact he never treated cases in London with these heroic doses. He could assure Dr. Guthrie that if he had the opportunity of treating any Scotch children for chorea in Scotland, he would strongly recommend him to give arsenic a chance, and he thought he would be very pleased with the result.

Dr. LEONARD GUTHRIE, in reply, said he had been extremely interested in the most valuable discussion which his paper had raised. As he began by saying, he had not anything new to state and he hoped to learn something for himself. He certainly had had the opportunity of hearing views put in the most interesting way by Dr. Cecil Wall, Dr. Lees, Dr. Sharpe and others. He did not know that he had very much to answer. After all, it must be admitted that these were questions of opinion. It was a very difficult thing to say when a drug did good and when it did not. In children the arthritic manifestations were very slight indeed. Probably if they put the child to bed and kept it there, warm and quiet, the rheumatism would subside, as it certainly did subside in out-patients who came with the history of what must have been articular rheumatism. Therefore, if they gave any drug in particular, and the symptoms subsided, they, of course, were naturally disposed to regard the recovery as entirely due to the drug. So far as aspirin and salicylate of soda were concerned, one was acid and the other alkaline; that was about the only difference, and he did not not see why one should have the preference over the other.

Dr. WALL: There is an acetyl radical in aspirin which makes a difference.

Dr. LEONARD GUTHRIE said they would regard it as a chemical difference. The active principles were the same. Let him do justice to salicylates. He had had a considerable experience with them personally. There was one kind of rheumatism which was very speedily relieved by salicylates—one characterized by a general condition of malaise, heaviness of head, usually associated with a strong tendency to yawn, and shooting, dull, aching pains all over the system. Two doses of 10 gr. or 15 gr. would drive those pains away. But let him speak of the other form of rheumatism to which many, he thought, were liable; those were cases called myositis, in which the rheumatism confined itself to the commencement or insertion of the muscles, usually of the neck, or about the shoulder. Now he had tried salicylates over and over again for that form of myositis. It had never been of the slightest use. The only thing which did good was the local application of leeches or dry cups. Drugs had no effect. Dr. Lees had told them, in a way which they could hardly refuse to accept, what he had seen; but he (the speaker) could not quite satisfy himself that the results of these large doses differed from results in other cases. If he could only convince himself, for instance, that if they gave 400 gr. of salicylate of soda a day to a child, it was cured of severe chorea in forty-eight hours, he would, of course, believe that there was something extraordinary in it. But Dr. Lees—he (the speaker) was sceptical, of course—had never quite satisfied him that the results were in any way commensurate with the largeness of the doses. Then there was a question as to the exact nature of chorea. Dr. Cecil Wall asked his opinion. He did not think that they were much at variance on the point; but after all, there again it was a matter of opinion. He regarded it as an affection of the higher centres, and he thought Dr. Cecil Wall did too; but the latter regarded it as a defective inhibition of the higher centres over the lower. He was not in the least prepared to say it was not so; uncontrollable impulses implied defective inhibition, but the only point he wished to make was that it was essentially an affection of the higher centres, in fact a psychosis. He did not know any psychosis which was affected by drugs, and that was one reason why he rather doubted the efficacy of any of the drugs mentioned in the case of chorea. Dr. Lees had spoken about the nodules; as to the prognosis he thought they were agreed on that point, but he believed that to some extent Dr. Lees thought that the nodules would disappear very rapidly under the use of these large doses of salicylate of soda. Well, he (the speaker) had seen them melt away over and over again without anything at all, so he should not be convinced, even if they did so, that they disappeared in consequence of the administration of the drug. Dr. Lees mentioned an interesting fact regarding the traumatic origin of chorea and parallel cases—cases of pneumonia. He (the speaker) agreed with Dr. Lees that the poison, or germ, was present at first, and that it was excited to action by any shock. One gentleman had spoken most enthusiastically as to the use of arsenic. He could only repeat that in this country he thought it was acknowledged that heroic doses of arsenic did not agree with children in hospital. Over and over again he had seen it do great harm. Only last year two cases of severe arsenical peripheral neuritis had been admitted to hospital. The only cases of chorea he had known to be fatal were those in association with severe pericarditis, or endocarditis, or septicæmia. He ought to say that he did believe in the use of the icebag in some cases of pericarditis, but in some cases it seemed rather too much for the child to bear. He spoke not so much of the pain as of the extreme restlessness and irritability which certain children suffered from, which made them infinitely worse in many cases. Sometimes they became extremely exacting and peevish. They would insist on having only one particular nurse to look after them. They would deliberately put themselves into frightful passions if they were thwarted in any way, and one was afraid that their lives would flicker out simply through passion and temper. He found that morphia soothed them and kept them quiet, and was, perhaps, better for them than any other form of treatment for the heart disease, which in those particular cases could only have a fatal issue.

INDEX

INDEX

(Prepared by Mr. A. L. CLARKE)

Abdomen, post-mortem appearances in case of congenital defect of muscular system, Path. 158, 160

——, wall of, sarcoma of, treatment by X-rays (W. Ironside Bruce), Electr. 60

——, see also *Intra-abdominal pressure*

——, see also *Tuberculosis* (abdominal)

Abdominal diseases, value of X-rays in diagnosis of obscure cases (Prof. E. Goldmann), Surg. 14

—— type of pneumonia, Med. 29, 30

Abductor paralysis, importance of correctness in designating cases of, Laryng. 39

ABERCROMBIE, PETER.—Further notes on a case of extensive cellulitis of the right side of the neck, Laryng. 10; discussion (p. 10): Mr. De Santi, Mr. Robinson, Mr. Horsford, Dr. Abercrombie (reply)

Abernethy, case of exostosis quoted, Path. 109, 110.

Abnormalities (congenital) in association with congenital pigmentation and atrophic scarring (H. G. Adamson), Derm. 9

ABRAHAM, P. S.—Case of favus in a mouse, shown by T. P. Beddoes, Derm. 64

ABRAHAMS, BERTRAM.—Case of myopathy, Clin. 24

——, discussion on multiple rheumatic nodules in adult, Clin. 14

Abrami and Burnet, results of cutaneous tuberculin reaction (alluded to), Path. 75

Abraxas grossulariata (currant-moth), var. *lacticolor*, inheritance of, Epid. 157, 158

Abscess, see under names of organs and regions affected

Acetabulum, perforation by head of femur, skiagram showing (H. Lewis Jones), Electr. 34

Acid, testing cavities of teeth for presence of, Odont. 119, 123

Acid-fast property, loss of, in bacteria, Path. 73

—— —— of bacteria in aural discharges, Otol. 104, 105, 106

—— —— of old nucleated squames in aural discharges, Otol. 99

Acidosis in pregnancy (J. B. Leathes), Path. 131

Acne, treatment by oral administration of vaccines, successful, Med. 243

Acromegaly, spurious ? (F. Parkes Weber), Clin. 104

—— —— ——, in patient suffering from exophthalmic goitre, associated with a congenitally high forehead (D. Walsh), Clin. 195

ADAMSON, H. G.—Case for diagnosis, Derm. 82; discussion (p. 83): Dr. Radcliffe Crocker

——, case of lupus erythematosus affecting the hands and feet only, Derm. 2

—— —— of nodular "ringed eruption," Derm. 81; discussion (p. 82): Dr. Radcliffe Crocker, Dr. Colcott Fox

—— —— of pustular ringworm of the horse ("conglomerate folliculitis") in a child, Derm. 63

—— —— of sclerodermia and leucodermia, Derm. 43

—— —— of sclerodermia (morphœa), Derm. 163; discussion (p. 163): Dr. Pernet, Dr. Crocker

—— —— of telangiectasis of the cheek, Derm. 44; discussion (p. 45): Dr. Radcliffe Crocker

—— —— of urticaria pigmentosa, Derm. 63; discussion (p. 64): Dr. Savill, Dr. Adamson (reply)

Adamson, H. G.—Circinate erythema of two years duration in a boy, Derm. 10
—— congenital pigmentation with atrophic scarring associated with other congenital abnormalities (shown for Dr. Ormerod), Derm. 9
—— discussion on case for diagnosis (lymphangioma circumscriptum), Derm. 49
—— —— —— of lichen spinulosus, Derm. 51
—— —— on granuloma annulare, Derm. 163
Addison's disease, resemblance of case of pigmentation with enlarged spleen and leucopenia to, Clin. 33
—— ——, symptoms of, in case of leucodermia (N. Dalton), Clin. 209
Adductor paralysis, importance of correctness in designating cases of, Laryng. 39
Adenitis, tuberculous, treatment of lupus following, by Roentgen rays combined with cauterization, Electr. 88
Adenoids, tumour removed from naso-pharynx fifteen weeks after removal of (E. F. Potter), Laryng. 9
—— (long-neglected), two cases illustrating effects of, on development of upper jaws and nasal septum (J. Donelan), Laryng. 84
Adenoma, solid and cystic, represented in cases of goitre removed by operation, Surg. 25
——, parathyroidal, Surg. 77
Adenotome (new) (H. J. Davis), Laryng. 81
Adrenalin and nitrite of amyl, combined injection, influence on blood-pressure, Therap. 34, 35
Adults, acquired atresia of introitus and vagina in (H. Macnaughton Jones), Obst. 325
——, multiple rheumatic nodules in (A. E. Garrod), Clin. 13 (F. H. Hawkins), Clin. 237
——, congenital heart disease in, cases (R. Murray Leslie), Clin. 34
Age-incidence in chorea, Therap. 74, 82
—— of diseases among school children, changes in, Epid. 57
—— —— —— —— ——, table showing, Epid. 56
—— of pyorrhœa alveolaris, Odont. 104
—— —— rheumatism, Therap. 68
—— —— rubella, Epid. 104, 109, 120
—— in small-pox epidemic in Trinidad, Epid. 242
Ague powders, Therap. 24
Aikin, W. A.—Discussion on case of a girl aged 17 with genuine baritone voice, Laryng. 61
Air-passages, upper, sarcoma of specimens from cases of, and cases simulating (W. Jobson Horne), Laryng. 100
Ala nasi, rodent ulcer of (A. W. Williams), Derm. 62
Alcoholism, fatal complication of pneumonia, Med. 28
Alimentary canal, post-mortem condition, in cases of lymphocythæmia, Clin. 168
Alkali, deficiency of, producing excess of ammonia in urine, Path. 140, 141
Alkaptonuria, causation of, theories respecting, Epid. 148, 162
——, connection of "presence and absence hypothesis" of relation between alternative pairs of characters with, Epid. 148, 149
—— and ochronosis, Clin. 57, 58
Allison, N., description of case of acquired coxa valga, quoted, Surg. 118
Alopecia areata, following small-spored ringworm of scalp (J. M. H. MacLeod), Derm. 89
Ammonia in urine, excess of, due to deficiency of alkali, Path. 140, 141
—— —— ——, high coefficient in subjects of severe vomiting of pregnancy, Path. 133
—— —— —— —— —— under ordinary and starvation diets, Path. 138, 139
—— —— ——, relation to excretion of lactic acid, Path. 131, 133
Amnion, human, development of, Obst. 277
Amputation (high) with cautery in treatment of cancer of cervix (H. R. Spencer), Obst. 31
Amyl, nitrite of, and adrenalin, combined injection, influence on blood-pressure, Therap. 34, 35,
Amyotonia congenita, cases of (J. Collier), Neur. 60
—— —— —— (W. E. Wynter), Clin. 48
Anæmia (cyanotic), case of (methæmoglobinæmia of twelve years' standing) (A. E. Garrod), Clin. 48

Anæmia (cyanotic), case referred to Committee, concluded to be one of intracorpuscular sulphæmoglobinæmia, Clin. 197, 198
—— (pernicious), treatment with antistreptococcus serum, Med. 197
—— (splenic), pigmentation with enlarged spleen and leucopenia ? Splenic anæmia (W. P. Herringham), Clin. 33
Anæsthesia (hypnotic), reasons for scientific importance of, Therap. 58
—— —— unreliable from uncertainty of induction, Therap. 58
—— (local), aid of, in partial grafting in after-treatment of radical mastoid operation (J. Stoddart Barr), Otol. 145, 148
Anæsthetics employed in operations for removal of goitre, Surg. 31, 77, 79
Anastomosis (arterio-venous), history of operation, Clin. 203, 204, 205
—— —— in treatment of senile gangrene (C. A. Ballance), Clin. 199
—— ——, special dexterity required for performance of, Clin. 206
Andalusian fowl, breeding of, simple illustration of Mendelism, Epid. 135, 137
ANDERSON, Miss L. G.—Sarcoma of the cervix (with report of Pathology Committee), Obst. 153; discussion (p. 155): Mrs. Boyd, Dr. Herbert Spencer
ANDREWES, F. W.—Discussion on etiology and epidemiology of typhoid fever, Epid. 223
ANDREWS, H. R., and MAXWELL, R. D.—Case of difficult labour, Obst. 138; discussion (p. 148): Dr. Champneys, Dr. W. S. A. Griffith, Dr. Herman, Dr. Eden, Dr. Herbert Spencer, Dr. Andrews (reply), Dr. Maxwell (reply)
Aneurysm, treatment by electrolysis, Electr. 124
—— (aortic), diagnosis confirmed by Roentgen ray examination, Electr. 52, 53
—— —— and paralysis of vocal cord, Electr. 52
—— (fusiform) of right common carotid artery (J. Hutchinson, jun.), Clin. 17, 21
—— of common femoral artery; excision of aneurysm and common femoral vein (C. H. Fagge), Clin. 232
Angina cruris, see *Angina pedis*
—— Ludovici, case after operation for (W. Stuart-Low), Laryng. 130
—— pectoris with aortitis and stenosis of coronary arteries (F. Parkes Weber), Clin. 114
—— —— (true), rarity in women, Clin. 118
—— pedis or angina cruris caused by chronic arteritis obliterans (F. Parkes Weber), Clin. 44, Neur. 50
Angioma of nose, pulsating (W. D. Harmer), Laryng. 142
—— of right tonsil (A. Wylie), Laryng. 109
——, see also *Telangiectases*
Angio-sarcoma of fourth ventricle, Neur. 27
Angle of inclination in coxa valga, Surg. 109, 110
Angle's classification of dento-facial irregularities, Odont. 141, 145
Aniline ions in treatment of lupus, Electr. 75, 76
Animal food, proportion of, lessened under era of cibiculture, Therap. 46, 47
Ankylosis, treatment by electrolysis, Electr. 122
Anopheles, destruction of, in prevention of malaria, Epid. 312, 316, 317
Anosmia, with dryness and crustings of the nose (C. Fox), Laryng. 13
Anti-autolytic powers of horse serum, Med. 246
Antibodies, production not always sufficient to produce immunity, Med. 196
——, presence in cerebrospinal fluid, Surg. 160, 162
Antimony, value in treatment of sleeping sickness proved experimentally, Therap. 26, 27, 28
Antiseptic fluids, injection of, combined with lumbar puncture in treatment of cerebrospinal meningitis, Surg. 161
Antitryptic power of horse serum, Med. 242
Antro-tympanic cavity, presence of cholesteatoma in, indicated by acid-fast property of non-nucleated squames in aural discharge, Otol. 99
Antrum, intermaxillary, existence of, in chimpanzee, Odont. 22
——, maxillary, development of, Odont. 20
—— —— ——, growth of maxilla closely related to, Odont. 20, 21

Antrum, maxillary, feeble development of, coexistent with great development of inferior meatus of nose, Odont. 22
—— ——, discharge from, cytological examination (J. M. Darling), Otol. 163
—— ——, large post-nasal polypus growing from, simulating nasal pharyngeal fibroma (J. Dundas Grant), Laryng. 102
—— ——, passage of pus from, to neck without passing through mastoid process (A. H. Cheatle), Otol. 24
—— ——, radical operation on, cases exemplifying simplification of after-treatment (W. Stuart-Low), Laryng. 58
—— ——, suppuration, chronic, operation through inferior meatus (J. Dundas Grant), Laryng. 30
Aorta, ruptured valve of (S. Taylor), Clin. 97
Aortitis in case of angina pectoris (F. Parkes Weber), Clin. 114
—— ——, etiology, Clin. 116
Aphonia, presence of, reason for removal of goitre, Surg. 25
—— (functional) cases of (J. Donelan), Laryng. 79
—— —— —— ——, (F. de Havilland Hall), Laryng. 77
Appendix, vermiform, concretions in, causing confusion in X-ray diagnosis of calculi, Electr. 29
Apprenticeship (medical), personal reminiscences of (W. Soper), Therap. 32
Arm, slinging of, to neck, with flexion of elbow to acute angle, in treatment of fractured humerus, Surg. 222-225, 230
—— (brawny) of breast cancer, lymphangioplasty for, two cases (W. S. Handley), Clin. 186
—— (right), case of tremor of, associated with epilepsy (F. E. Batten), Neur. 54
Arm-baths, medium for electrical applications, Electr. 115
ARMOUR, D.—Cervical caries, operation and recovery, Neur. 64; discussion (p. 65): Dr. F. Buzzard
——, discussion on case of disease of cervical vertebræ with pressure symptoms, Neur. 14
—— —— on multiple neuro-fibromata and spastic paraplegia, Neur. 63
Army (British) mortality from malaria and yellow fever in West Indies (1780-96), Epid. 304, 305
ARNIM, Major.—Discussion on plague, Epid. 87
Arsenic in treatment of acute stage of chorea, Therap. 77, 80, 87
Arteries, changes in, during process of arterio-sclerosis, Therap. 34, 35
——, disease of, experimental production, Therap. 33
——, isolators of pathological processes, Surg. 3
——, middle coat of, principal seat of changes in arterio-sclerosis, Therap. 37
—— (coronary), stenosis of complicating angina pectoris with aortitis (F. Parkes Weber), Clin. 114
——, see also *Anastomosis*, arterio-venous
Arterio-sclerosis, Therap. 33
——, deficiency in thyroid secretion not connected with, Therap. 34
——, etiology of, Therap. 34, 35
——, fatty degeneration in, Therap. 33
——, irregular heart's action under, digitalis without effect on, Therap. 30
——, muscle fibres essential seat of, Therap. 36
——, production of, Therap. 33, 34
—— ——, by drugs having power of raising blood-pressure, Therap. 34
Arteritis obliterans of lower extremity with intermittent claudication ("angina cruris") (F. Parkes Weber), Clin. 44, Neur. 50
—— —— (chronic), cause of intermittent claudication in lower extremity (angina pedis or angina cruris) (F. Parkes Weber), Clin. 44, Neur. 50
—— —— of right upper extremity associated with malformation of both clavicles (L. G. Guthrie), Neur. 101
Artery (carotid, right common), fusiform aneurysm of (J. Hutchinson, jun.), Clin. 17, 21

Artery (coronary, right), embolism of, causing death in case of pneumonia complicated by endocarditis (case referred to in discussion), Clin. 118
—— (femoral), excision of parts of femoral artery and vein for recurrent sarcoma (C. H. Fagge), Clin. 231
—— —— (common), aneurysm of; excision of aneurysm and common femoral vein (C. H. Fagge), Clin. 232
Arthrectomy twice performed for tuberculous synovitis of knee-joint, movable joint resulting (D. Drew), Clin. 10
Arthritis complicating pneumonia, Med. 12
——, recovery from, under treatment by antidiphtheritic serum, Med. 107
ARTHUR, DAVID.—Discussion on cauterization as an adjuvant to radiotherapy, Electr. 89
——. discussion on diagnostic value of Röntgen rays in some diseases of chest, Electr. 44
—— —— —— electrical treatment of atonic conditions of digestive system. Electr. 108
Arytænoid, swelling of (A. Wylie), Laryng. 60
—— (right), swelling of, with loss of movement of right vocal cord (W. H. Kelson), Laryng. 108
—— cartilage, necrosis of (H. Barwell), Laryng. 65
Asafœtida, carminatived, value in dyspepsia with flatulence. Therap. 19
Ascites accompanying hepato-splenomegaly (H. Batty Shaw), Clin. 56
——, cure by permanent drainage through femoral ring (W. E. Wynter), Clin. 49
—— in case of myxœdema, Clin. 27
—— treatment by paracentesis twenty-five times in fifteen months; patient well seven years later (F. H. Hawkins), Clin. 190
Aspergillosis, coexistence with tuberculosis in birds, Path. 20
Aspergillus niger, Otol. 116
Aspiration under X-rays, pneumothorax treated by (J. Fawcett), Clin. 38
Aspirin in treatment of chorea, Therap. 77, 79, 80, 81
—— —— —— acute rheumatism, Therap. 70, 84, 85
Astereognosis, association of gross lesion of post-central gyrus with (P. Stewart), Clin. 220
Asylums, typhoid "carriers" in, Epid. 212, 213
Ataxy and involuntary movements, pathological changes in nervous system of dog suffering from (F. E. Batten and G. Holmes), Neur. 22
—— (cerebellar), two cases of (W. Harris), Neur. 52
—— (locomotor) (G. Ogilvie), Neur. 93
—— —— in chorea, Therap. 76
—— —— ——, treatment by exercises, Therap. 76
Atonic conditions of digestive system, treatment by electricity (R. Morton), Electr. 101
Atoxyl, value in treatment of sleeping sickness, Therap. 26
Atresia of introitus and vagina in adult, acquired (H. Macnaughton Jones), Obst. 325.
—— of naso-pharynx (C. Potter), Laryng. 15
——, partial and progressive, of naso-pharynx and oro-pharynx in case of complete closure of anterior nares (W. Stuart-Low), Laryng. 56.
—— (traumatic) of vagina treated by Cæsarean hysterectomy (A. Routh), Obst. 1
Atrophy (cerebellar) (F. E. Batten), Clin. 18
—— (macular) of scalp (pseudo-pelade of Brocq) (A. Whitfield), Derm. 60
—— (muscular), with delayed thermal sensation (Mr. Kilgour for Dr. L. Guthrie), Neur. 10
—— (peroneal), two cases (W. Harris), Neur. 54
Auditory canal, secondary involvement of, in case of epithelioma of right auricle (H. Tod), Otol. 16
AULD, A. G.—Discussion on vaccine treatment by the mouth, Med. 245
Aura, epileptic, Med. 94
Auricle, chronic eczema of both auricles and meatuses with unusual degree of hyperplasia (E. Furniss Potter), Otol. 132
—— (left), epithelioma of, with secondary involvement of auditory canal (H. Tod), Otol. 16
—— (cardiac) of tortoise, tonus curves from, Med. 254, 255

AUSTIN, L. J., and GRUNBAUM. OTTO.—Some experiences with the tuberculin ophthalmic reaction, Path. 74
Autograph letter of Johann Nepomuk Czermak (W. Jobson Horne), Laryng. 83
Aveling's repositor, see *Elastic pressure*, continued (Aveling's method)
Azam, Dr., first French investigator of hypnotism, Therap. 53

BABER, E. CRESSWELL.—Discussion on atresia of naso-pharynx, Laryng. 16
——, discussion on case of complete closure of the anterior nares, with partial and progressive atresia of the naso-pharynx and oro-pharynx, Laryng. 57
—— —— on case of ulceration and destruction of epiglottis, Laryng. 34
—— —— on extreme form of septal deflection, Laryng. 6
—— —— on neoplasm of right tonsil, Laryng. 141
—— —— on specimen of large post-nasal polypus, Laryng. 122
—— —— on sudden deafness in case of syphilis, Otol. 8
——, microscopic specimen from a case of ulceration of the inferior turbinated body, Laryng. 41
Bacillus butyricus, Otol. 105, 116
—— —— and fœtor in aural discharges, Otol. 96
Bacillus coli communis, infective capabilities of, Epid. 216
—— —— —— infection, phagocytosis of leucocytes from different cases of, Med. 185
—— —— —— substituted for melanin, amount of phagocytosis, Med. 187, 188, 193
—— diphtheroid, associated with case of acute suffocative catarrh of Laennec (S. West), Med. 151, 153
—— *enteritidis*, Gaertner, meat poisoning by, poisoning from eating shell-fish comparable to, Path. 82
—— —— ——, infection in infants (F. E. Batten and J. G. Forbes), Clin. 81
—— *fusiformis*, throat organism in chronic aural discharge, Otol. 115
—— *influenzæ* in aural discharges, Otol. 112
—— *paratyphosus*, abscess of bone caused by an intermediate bacillus (P.) allied to (F. G. Bushnell), Path. 79
—— —— —— —— probable value in diagnosis of paratyphoid fever, Path. 81
—— (*P.*) intermediate, allied to *Bacillus paratyphosus*, bacteriological report on, Path. 79
—— of plague, easily destroyed, Epid. 74
—— *proteus vulgaris*, Otol. 116
—— —— —— and fœtor in aural discharges, Otol. 96
—— —— ——, throat organism in chronic aural discharge, Otol. 112, 115
—— *pyocyaneus* producing tints in aural discharges, Otol. 97
——, question of isolation, in acute ulcerative colitis, Med. 163, 164, 165, 167
—— *subtilis* in aural discharges, Otol. 112
—— *tuberculosis*, old, non-nucleated squames in aural discharges mistaken for, Otol. 99
—— —— in chronic aural discharge, Otol. 116
—— ——, see also under *Tubercle bacillus*
—— *typhosus*, "carriers" of, transmission of typhoid fever by, Epid. 175-191; see also under "*Carriers*"
—— ——, isolation from water supplies, Epid. 225
—— ——, methods of isolation, Epid. 184
—— ——, relation to typhoid fever (W. H. Hamer), Epid. 204-218
—— —— —— ——, Epid. 221, 224
—— —— —— —— —— ——, review of difficulties respecting, Epid. 215-218, 223
—— ——, inoculation with, conferring protective value, Epid. 223
Bacilli, dead, absorption of, producing immunity, Med. 195, 196
Back, muscles of, post-mortem appearances in case of congenital defect of muscular system, Path. 160
Bacon, well-cooked, as article of diet in irritable dyspepsia, Therap. 15, 20
Bacteria, differentiation amongst members of any group of, functional rather than morphological, Path. 72

Bacteria, differentiation amongst, in aural discharges, Otol. 102-113
—— —— —— acid-fast, Otol. 105, 106
—— —— —— ——, clostridia, Otol. 105
—— —— —— ——, streptothricial, Otol. 105
—— —— —— ——, rod forms, Otol. 105
—— —— —— acute, Otol. 101
—— —— ——, best classification, Otol. 119, 120
—— —— ——, chronic, Otol. 103
—— in peritoneum, Path. 108
—— loss of acid-fast quality in, Path. 73
—— transport to internal organs of healthy animals, Path. 108
Bacterial origin, probable, of swelling of callus formed after fractures, Path. 1[illegible]
Bacteriuria in women, treatment by ionic medication, Electr. 135
Badcock, J. H.—Discussion on case of extensive necrosis of mandible, Odont. 32
—— —— discussion on treatment of pyorrhœa, Odont. 65
Bainbridge, F. A.—Discussion on acidosis in pregnancy, Path. 145
Baker, A. E.—Discussion on dental uses of paraform, Odont. 43
——, Arthur W. W.—A difficult extraction, Odont. 130; discussion (p. 138): Mr. Howard Mummery, Mr. Montagu Hopson, Mr. Dowsett, Mr. Baldwin, Dr. Arthur Baker (reply)
—— ——, discussion on classification of dento-facial irregularities, Odont. 144
—— —— on diffuse fibromata of gums, Odont. 128
Baldness, advanced keratosis follicularis associated with (J. M. H. MacLeod and E. Treacher Collins), Derm. 27
Baldwin, H.—Discussion on classification of dento-facial irregularities, Odont. 145
—— discussion on dental uses of paraform, Odont. 42
—— —— on difficult extraction, Odont. 140
—— —— on diffuse fibromata of gums, Odont. 128
—— —— on growth of jaws, Odont. 25
—— —— on heredity and dental disease, Odont. 123
—— —— on teeth of fossil fishes, Odont. 16
—— —— on treatment of pyorrhœa, Odont. 67
Ball, J. B.—Case of pulsating growth in the left external auditory meatus, Otol. 32; discussion (p. 33): Dr. McBride, Mr. Waggett, Dr. Milligan, Mr. Fagge, Dr. W. Hill, Dr. Ball (reply)
—— discussion on atresia of naso-pharynx, Laryng. 16
—— —— on bilateral swelling of the septum nasi, Laryng. 93
—— —— on bleeding polypus of septum, Laryng. 29
—— —— on case after operation for angina Ludovici, Laryng. 130
—— —— —— of chronic suppuration of maxillary antrum, Laryng. 32
—— —— —— of congenital laryngeal stridor, Laryng. 110
—— —— —— of congestion of right vocal cord, Laryng. 18
—— —— —— of fibrous constriction of trachea, Laryng. 35
—— —— —— of intranasal hypertrophy, Laryng. 47
—— —— —— of lupus of epiglottis, Laryng. 106
—— —— —— of mucocele of frontal sinus, Laryng. 72
—— —— —— of nasal obstruction, Laryng. 75
—— —— —— of partial occlusion of both anterior nares by a congenital cutaneous web, Laryng. 66
—— —— —— of tumour of right thyroid lobe, Laryng. 10
—— —— on cases after the radical maxillary antrum operation, exemplifying simplification of the after-treatment, Laryng. 58
—— —— on four cases illustrating surgery of sphenoidal sinus, Laryng. 129
—— —— on effects of long-neglected adenoids on development of upper jaws and nasal septum, Laryng. 84

BALL, J. B.—Discussion on extreme form of septal deflection, Laryng. 6
—— —— on fistulous opening in middle line of neck, Laryng. 4
—— —— on hysteria with very unusual laryngeal manifestations, Laryng. 135
—— —— on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 103
—— —— on lupoid ulcer of floor of vestibule of nose, Laryng. 110
—— —— on paresis of right vocal cord, Laryng. 2
—— —— on septal deflection in phthisical patient, Laryng. 111
—— —— on specimen of large post-nasal polypus removed from boy, Laryng. 122
—— —— on suppurating hæmatoma of septum nasi, with fistulous opening in gingivo-labial recess, Laryng. 93
—— —— on tumour of thyroid gland, Laryng. 47
BALLANCE, C. A., M.V.O.—Case of arterio-venous anastomosis for senile gangrene, Clin. 199
—— discussion on case of hair-ball in stomach, Clin. 214
—— —— on case of trigeminal neuralgia treated by excision of Gasserian ganglion, Clin. 22
—— —— on keloid following mastoid operation, Otol. 22
—— —— on lateral sinus thrombosis, Otol. 140
—— —— on lumbar puncture in otitic meningitis, Surg. 163
—— —— on mastoid operation, Otol. 147
—— and BARLOW, Sir THOMAS.—Case of rupture of the upper cord of the brachial plexus at birth, Clin. 215; discussion (p. 219): Dr. Poynton, Mr. C. H. Fagge, Mr. Lockhart Mummery, Mr. Ballance (reply)
BALY, C. F. P.—Discussion on treatment of children from dental aspect, Odont. 76
Bandaging above and below in ununited fractures, Surg. 237
Bantams, see *Rosecomb* bantams.
Bárány, observations of, on thermal nystagmus referred to, Neur. 82
Baritone voice (genuine) in girl aged 17 (C. Horsford), Laryng. 61
Barium sulphate (coarse), mixture of, with vaseline, for shielding parts of scalp not to be exposed in treatment of ringworm by X-rays (A. Whitfield), Derm. 61
BARKER, ARTHUR E. J.—Meningitis complicating otitis media, with lumbar puncture, Clin. 74
—— on the possible use of lumbar puncture in the treatment of otitic meningitis (with references), Surg. 143-162; discussion (p. 163): Mr. Charles A. Ballance, Dr. Frederick E. Batten, Mr. Barker (reply)
BARLOW, Sir T., Bt., K.C.V.O.—Discussion on amyotonia congenita, Clin. 49
—— discussion on case of angina pectoris with aortitis, Clin. 119
—— —— —— of cerebellar tumour with proptosis, Clin. 128
—— —— —— of enlarged spleen with polycythæmia, without cyanosis, Clin. 60
—— —— —— of fatal purpura hæmorrhagica, Clin. 122
—— —— —— of leucodermia with symptoms of Addison's disease, Clin. 212
—— —— —— of methæmoglobinæmia, Clin. 48
—— —— —— of myxœdema with optic atrophy, Clin. 195
—— —— —— —— with unusual features, Clin. 29
—— —— —— of multiple hereditary developmental angiomata, Clin. 66
—— —— on cases of lymphocythæmia, Clin. 177
—— —— on multiple rheumatic nodules in adult, Clin. 14
—— —— on splenic polycythæmia, with cyanosis, Clin. 43
——, presidential address at opening meeting of Clinical Section, Clin. 1
—— and BALLANCE, C. A.—Case of rupture of the upper cord of the brachial plexus at birth, Clin. 215; discussion (p. 219): Dr. Poynton, Mr. C. H. Fagge, Mr. Lockhart Mummery, Mr. Ballance (reply)
BARR, J. STODDART.—Notes on partial grafting, with the aid of local anæsthesia, in the after-treatment of the radical mastoid operation, Otol. 145, 148; discussion (p. 146): Dr. Syme, Mr. A. L. Whitehead, Dr. Kerr Love, Mr. C. A. Ballance, Dr. Milligan, Dr. Thomas Barr, Dr. Logan Turner (reply), Dr. Porter (reply), Dr. Stoddart Barr (reply)

Barr, Thomas.—Discussion on lateral sinus thrombosis, Otol. 139

——, discussion on partial grafting in after-treatment of radical mastoid operation, Otol. 148

Barwell, Harold S.—Case of a man on whom thyrotomy had been performed one year and nine months before for tuberculous laryngitis, Laryng. 7; discussion (pp. 7, 8): Dr. de Havilland Hall, Dr. Dundas Grant, Mr. Herbert Tilley, Dr. W. Hill, Dr. Jobson Horne, Mr. Barwell (reply)

—— case of extreme form of septal deflection, Laryng. 5; discussion (p. 6): Dr. Dundas Grant, Dr. Scanes Spicer, Mr. De Santi, Mr. Stuart-Low, Mr. Clayton Fox, Dr. StClair Thomson, Dr. Donelan, Mr. Cresswell Baber, the President (Dr. Ball), Mr. Barwell (reply)

—— case of necrosis of the arytænoid cartilage, Laryng. 65; discussion (p. 65): Mr. C. Fox, Mr. De Santi, Mr. Barwell (reply)

—— discussion on case of bilateral frontal sinusitis, Laryng. 26

—— —— —— of chronic empyema of left frontal sinus treated by simplified method of Killian's operation, Laryng. 36

—— —— —— of epithelioma of left tonsil, faucial pillar, and uvula, Laryng. 30

—— —— —— of gumma of larynx, Laryng. 20

—— —— —— of laryngeal disease for diagnosis, Laryng. 12

—— —— —— of laryngeal paralysis, Laryng. 107

—— —— —— of laryngeal ulceration, Laryng. 23

—— —— —— of lupus of the larynx and tonsil, Laryng. 78

—— —— —— of swelling of right arytænoid, Laryng. 108

—— —— —— of tumour of vocal cord, Laryng. 9

—— —— —— of ulceration and destruction of epiglottis, Laryng. 34

—— —— on effects of long-neglected adenoids on development of upper jaws and nasal septum, Laryng. 86

—— —— on lupoid ulcer of vestibule of nose, Laryng. 110

—— —— on malignant growth behind cricoid cartilage, Laryng. 111

—— —— on new adenotome, Laryng. 81

—— —— on proptosis due to distension of frontal sinus, Laryng. 50

—— —— on septal deflection in phthisical patient, Laryng. 111

——, symmetrical nodules on the cords of a boy aged 9, Laryng. 18; discussion (p. 19): Mr. Herbert Tilley, Mr. Clayton Fox, Sir Felix Semon

Bateson, W., investigations respecting colour-blindness quoted, Epid. 157

Batten, F. E.—Case of cerebellar atrophy, Clin. 18

—— case of tremor of the right arm associated with epilepsy, Neur. 54; discussion (p. 56): Dr. E. Jones, Dr. Beevor, Dr. G. Holmes, Dr. Batten (reply)

—— discussion on case of amyotonia congenita, Neur. 61

—— —— on lumbar puncture in otitic meningitis, Surg. 164

—— —— on mechanism of nystagmus, Neur. 80

—— —— on occurrence of muscle spindles in ocular muscles, Neur. 87

—— —— on tumours of fourth ventricle, Neur. 37

—— and Forbes, J. G.—Note on two cases of Gaertner infection (*Bacillus enteritidis*) in infants, Clin. 81

—— and Holmes, Gordon.—The nervous system of a dog which suffered from ataxia and involuntary movements, Neur. 22; discussion (p. 26): the President, Dr. Ferrier, Dr. Purves Stewart, Sir Victor Horsley

Batten, G. B.—Discussion on electrical treatment of atonic conditions of digestive system, Electr. 109

—— —— on ionic medication, Electr. 80

Battle, W. H., and Shattock, S. G.—A remarkable case of diffuse cancellous osteoma of the femur following a fracture in which similar growths afterwards developed in connection with other bones, Path. 83-114

Baynes, Donald.—Discussion on ionic medication, Electr. 78

Baynes, Donald.—Discussion on treatment of leukæmia, exophthalmic goitre and malignant disease by X-rays, Electr. 61

Bazin's disease (erythema induratum) case of (E. G. Graham Little), Derm. 27

—— —— —— ——, case in which positive ophthalmo-tuberculin reaction has been obtained (J. M. H. MacLeod), Derm. 31

Beckett-Overy, Harry.—Discussion on intra-uterine death of syphilitic fœtus in six consecutive pregnancies, Obst. 171

—— discussion on menstruation and the calcium metabolism, Obst. 314

Beddard, A. P.—Discussion on acidosis in pregnancy, Path. 144

Bedding, source of typhoid infection, Epid. 222

Beddoes, T. P.—Case of favus in a mouse (shown for Dr. Abraham), Derm. 64

——, discussion on case of gonorrhœal warts, Electr. 64

Bees, explanation of, ancient belief regarding origin of, Path. 151

Beevor, C. E.—Case of myatonia congenita, Neur. 1

—— case of toxic affections of muscles and lower neurones, Neur. 2; discussion (p. 3): Dr. J. S. Collier, Dr. S. A. K. Wilson, Dr. Campbell Thomson, Dr. Wilfred Harris, Dr. Gordon Holmes, Dr. Ormerod, Dr. Beevor (reply)

—— discussion on case of amyotonia congenita, Neur. 62

—— —— on case of dog suffering from ataxia, Neur. 26

—— —— —— of isolated paresis of right serratus magnus, Neur. 10

—— —— —— of slowly progressive hemiplegia, Neur. 6

—— —— —— of tremor of the right arm associated with epilepsy, Neur. 56

—— —— on hemiplegia with involuntary movements, Neur. 56

—— —— on pathology of epilepsy, Med. 113

—— —— on pathology and treatment of constipation, Med. 148

—— —— on tumours of fourth ventricle, Neur. 37

—— —— on two cases of tabes, Neur. 66

——, introductory remark at opening meeting of Neurological Section, Neur. 1

Bell, W. Blair.—Menstruation and its relation to the calcium metabolism (with references), Obst. 291; discussion (p. 313): Dr. Pollock, Dr. Amand Routh, Dr. Beckett-Overy

Benham, R. F., on association of arrest of heart's action with tonico-clonic spasms; quoted, Med. 74

Bennett, F. J.—Discussion on growth of jaws, Odont. 24

—— discussion on heredity and dental disease, Odont. 119, 123

—— —— on movements of mandible, Odont 96

—— —— on teeth of fossil fishes, Odont. 16

—— —— on treatment of children from dental aspect, Odont. 73

——, resolution to appoint subcommittee of Odontological Section to consider and report on best method of arriving at exact knowledge of causes of dental caries, Odont. 73, 78

Bennett, Norman G.—Contribution to the study of the movements of the mandible, Odont. 79; discussion (p. 96): Mr. Rushton, Mr. F. J. Bennett, Mr. Gabell, Mr. E. Lloyd-Williams, Mr. Dowsett, Dr. Sim Wallace, Mr. N. G. Bennett (reply)

——, discussion on clinical notes on pyorrhœa alveolaris, Odont. 105

—— —— on growth of jaws, Odont. 25

—— —— on heredity and dental disease, Odont. 121

Bernheim, hypnotic researches of, inspired by Liébault, Therap. 54

Bernstein, Julius.—Discussion on fatal illness from status lymphaticus, Clin. 79

——, pathological report on Mr. A. Evans's specimens of diffuse fibromata of gum, Odont. 127

Berry, James.—An analysis of 274 additional cases of removal of goitre by operation, Surg. 21-75; discussion (p. 76): Mr. Warrington Haward, Sir Victor Horsley, Mr. W. G. Spencer, Mr. Herbert Paterson, Mr. Berry (reply)

Bethe's method of staining described, Odont. 50

Bezold's mastoid empyema, discharging to pharynx (W. S. Syme), Otol. 125

Bibliographies.—Abscess of bone, Path. 82

——, acidosis in pregnancy, Path. 143

Bibliographies, arterio-venous anastomosis for gangrene, Clin. 207
——, aural discharges, clinical pathology, Otol. 117
——, cardiac muscle, tone of, Med. 270
——, coxa valga, Surg. 140
——, cystic tumour of suprarenal body, Surg. 217
——, dermoid, ovarian, Obst. 117
——, endocarditis, tuberculous, Path. 123
——, epilepsy, pathology of, Med. 110
——, gouty deposit, cases of, Clin. 230
——, grape-like sarcoma of the cervix uteri, Obst. 259
——, hæmorrhage into pons Varolii in eclampsia of pregnancy, Obst. 79
——, hemihypertrophy, Clin. 64
——, labyrinthitis, operative surgery of, Otol. 73
——, lymphocythæmia in early life, Clin. 177
——, lumbar puncture in otitic meningitis, Surg. 162
——, menstruation and the calcium metabolism, Obst. 312
——, typhoid carriers, Epid. 191
Biernacki, J.—Discussion on pathology of epilepsy, Med. 115
Biffen, Mr., experiments respecting immunity to disease in wheat quoted, Epid. 140
Bigeminal action of digitalis, Med. 262
Billington, W., case of macroglossia neurofibromatosa, quoted, Path. 8
Birds, cause of spread of molluscum contagiosum, Derm. 172
——, human tubercle bacillus not converted into avian by inoculation into birds, Path. 70, 71
——, labyrinth of, stereoscopic photographs illustrating (A. A. Gray), Otol. 164
——, opsonic index raised above normal in birds actively tuberculous, Path. 65
——, parasitic invasion in, simulating tuberculosis, Path. 21
——, serum of, inhibitive action upon leucocytes of normal human blood, Path. 68
——, sexual gland in, indications of hermaphroditic condition of, Path. 7
——, tuberculous material from, feeding of pigeons with, Path. 34
—— —— ——, feeding of rhesus monkey with, Path. 53
—— —— ——, inoculation of guinea-pigs with suspensions of, Path. 50
—— —— —— ——, arrest and retrogression of process, Path. 52
——, tuberculous organs of, salt suspensions inoculated into pigeons, experiments and results, Path. 46
——, tuberculosis in, abdominal commonest form, Path. 17
—— —— involvement of thyroid and thymus glands, Path. 18
—— —— matting of abdominal and thoracic viscera, Path. 20
—— —— pulmonary, associated with abdominal form, Path. 18
—— —— see also *Tuberculosis* (avian)
Birth, rupture of upper cord of brachial plexus at (Sir T. Barlow and C. A. Ballance), Clin. 215
Bismuth: liquor bismuthi combined with salicylate of soda and bicarbonate of soda, value in rheumatism, Therap. 86
—— valuable in diminishing gastric secretion, Therap. 5
—— —— in treatment of gastric hyperæsthesia, Therap. 7, 10, 11
—— breakfasts in cases of chronic constipation, before examination under X-rays, Med. 120-147
Blacker, G. F.—Discussion on case of fibroid tumour complicating pregnancy, Obst. 319
——, discussion on development of early human ovum, Obst. 277
—— —— on pubiotomy, Obst. 30
Bland-Sutton, John.—A case of pregnancy complicated by a large cervical fibroid: subtotal hysterectomy at the fourth month, Obst. 17; discussion (p. 18): Dr. H. R. Spencer, Dr. Arthur Giles, Mr. Bland-Sutton (reply)
——, case of sarcoma of the uterus, Obst. 233 (with report of Pathology Committee, p. 235); discussion (p. 236): Dr. Amand Routh, Dr. Macnaughton Jones, Dr. Herbert Spencer

BLAND-SUTTON, JOHN.—Two examples of the encapsulation of sterile fluid in connection with the Fallopian tube and the ovary, Obst. 229
Blood, circulation of, in malignant growths, Surg. 5
——, coagulability of, increased by hypodermic administration of horse serum or fresh anti-diphtheritic serum, Med. 198
——, condition of, in cases of lymphocythæmia, Clin. 135, 140, 143, 144, 147, 150, 153, 154, 156, 157, 160, 174, 175
—— from subject of infective disease, addition of melanin to ; result respecting phagocytosis of melanin, Med. 173, 177
——, lactic acid in, in eclampsia, Path. 131
——, human, normal, leucocytes of, inhibitive action of birds' serum upon, Path. 68
Blood-calcium, metabolism of, relation to egg-laying, Obst. 295, 296
—— —— relation to menstruation, Obst. 295, 297-300
—— —— —— —— ——, experiments and observations on rabbits respecting, Obst. 301-305
Blood-cells, amounts of phagocytosis of melanin on presentation of melanin to washed cells of patient, and to washed normal cells in immune serum, Med. 184-191
——, production *in vitro* of opsonin by patient's cells in patient's serum, Med. 191-194
——, separation of, for phagocytosis observations, Med. 171
—— (immune) must work in immune serum to form correct estimate of patient's hæmo-phagocytic resistance, Med. 190, 191, 194
Blood-clot, cerebral intravascular, as cause of epilepsy, Med. 93
Blood-corpuscles, red, over-production of, in bone marrow, pathological feature of polycythæmia, Clin. 41
Blood-cyst of suprarenal body, Surg. 201-217
——, see also under *Suprarenal body*, cystic tumour of
Blood-letting, value of, Therap. 32
Blood-pressure, effect of dumb-bell exercise on, Therap. 36
——, effect of smoking on, Therap. 36
—— in epilepsy, Med. 83
—— (rise in), production of arteriosclerosis by all drugs possessing power of causing, Therap. 34
—— ——, proved conclusively to be sole cause of arterial disease, Therap. 35
Blood-serum, separation of, for phagocytosis observations, Med. 172
Blood-vessels, multiplication in malignant growths, purpose of, Surg. 10
BOKENHAM, T. J.—Discussion on electrical treatment of atonic conditions of digestive system, Electr. 108
BOLTON, Dr.—Discussion on ionic medication, Electr. 78
BOND, J. W.—Case of malignant growth situated behind the cricoid cartilage, Laryng. 111 ; discussion (p. 111) : Mr. Barwell, Dr. FitzGerald Powell
BOND, W. A.—Discussion on etiology and epidemiology of typhoid fever, Epid. 226
Bone, abscess of, caused by an intermediate bacillus (P.) allied to *B. typhosus*, Path. 79
——, destructive changes in, occurring in gout, cases showing (J. B. Burt), Clin. 223
——, invasion of wisdom tooth by, Odont. 135
——, plantation of small fragments of, between ends, in treatment of ununited fracture (Rushton Parker), Surg. 232
——, regeneration of, Surg. 235, 236
Bone-marrow as primary seat of disease in acute lymphocythæmia, theory respecting, Clin. 169
—— ——, over-production of red corpuscles in, pathological feature of splenic polycythæmia, Clin. 41
Bones, disease of, in goats, Path. 104
——, invasion of, wide-seated, in case of sarcoma of rectum, Path. 124
BONNEY, W. VICTOR.—Discussion on removal of cancerous uterus and glands by Wertheim's method, Obst. 64
BOSANQUET, W. CECIL.—Statistics of complications of pneumonia from Charing Cross Hospital, Med. 71

Bowen, W. H.—Discussion on foreign body in ear, Otol. 7
Box, C. R.—Case of convulsive attack accompanying pulmonary embolism, quoted, Med. 97
——, discussion on congenital heart disease in adults, Clin. 37
Boyd, Mrs. F.—Discussion on bilateral tuberculous salpingitis, Obst. 262
—— discussion on case of uterine fibro-myoma, Obst. 77
—— —— on myomectomy during pregnancy, Obst. 138
—— —— on removal of cancerous uterus and glands by Wertheim's method, Obst. 65
Brachial plexus, neuritis of (W. P. Herringham), Clin. 31
—— ——, rupture of upper cord of, at birth (Sir T. Barlow and C. A. Ballance), Clin. 215
Brachydactyly, case of, illustrating Mendelism, Epid. 141
Bradford, J. Rose, F.R.S., and Shaw, H. Batty, cases of lymphocythæmia quoted, Clin. 131
Bradycardia, association with minor epilepsy, Med. 90
—— and other cardiac affections; association of syncopal and convulsive seizures with, Med. 73
Braid, James, originator of hypnotism, Therap. 51
—— substituted term "hypnotism" for that of "mesmerism," Therap. 52
Braillon and Jousset, on the causation of endocarditis by tubercle bacilli, Path. 122
Brain, abscess of, complicating pneumonia, Med. 11
—— (base of), see *Pons Varolii*
——, post-mortem condition of, in case of congenital defect of muscular system, Path. 158
——, tumour of motor area of, operated upon (H. C. Thomson), Neur. 96
——, vasomotor spasm in, a cause of epilepsy, Med. 87
——, see also *Frontal lobe*, *Gyrus*, *post-central*, *Mid-brain*
—— —— *Nucleus*
—— —— *Ventricle*
Bramwell, J. Milne.—Hypnotism, Therap. 49; discussion (p. 61): Dr. J. Gray Duncanson, Dr. E. B. Sherlock, Dr. Bramwell (reply)
Branchial sinus leading into external auditory meatus, excision (C. H. Fagge), Otol. 90
Braune's retraction (contraction) ring of uterus, Obst. 144-146, 148, 149, 150
Breast, cancer of, lymphangioplasty for brawny arm occurring with (W. S. Handley), Clin. 186
Breast-bone of chicken impacted in larynx for nearly forty-eight hours (F. A. Rose), Laryng. 5
Breast-feeding, important in prevention of future dental caries, Odont. 37
Breda fowl, crossing of, with comb-bearing fowl, illustration of nature of alternative pairs of characters, Epid. 148
Brentry Certified Inebriate Reformatory, Bristol, outbreak of typhoid fever at, caused by "carrier," Epid. 177
Breton, on the tuberculin ophthalmic reaction, alluded to, Path. 75
"Bridle" formation in larynx (H. Tilley), Laryng. 16
Briggs, Henry.—Discussion on removal of cancerous uterus and glands by Wertheim's method, Obst. 66
—— —— on the supports of the pelvic viscera, Obst. 60
—— —— —— —— ——, Obst. 221
Brincker, J. A. H., and Richards, H. Meredith.—The potential dangers of water derived from wells in the chalk, Epid. 191
Briscoe, J. Charlton, and Wiltshire, H. W.—Statistics of complications of pneumonia from King's College Hospital (1897-1906), Med. 60
Bristol: see *Brentry* Certified Inebriate Reformatory; *Grove* House Home for Girls, Brislington
Bristowe, J. S., on association of convulsive and epileptic seizures with bradycardia, quoted, Med. 73
Broadbent, Sir John, Bt.—Discussion on ruptured aortic valve, Clin. 98
Bromide in treatment of acute stage of chorea, Therap. 76, 81
——, eruption following administration of, occurring in mother: transmitted to breast-fed child, Derm. 73
Bronchitis (capillary), diagnosis from acute suffocative pulmonary œdema, Med. 153, 156

Broncho-pneumonia, treatment by antidiphtheritic serum, hypodermic administration, Med. 197
—— treatment by fresh horse serum administered by mouth, Med. 197
—— (primary), close alliance to suffocative catarrh, Med. 153, 156
—— (secondary), diagnosis from acute suffocative pulmonary œdema, Med. 153, 156
Bronner, Adolph.—Discussion on case of gumma of larynx, Laryng. 20
—— —— on cases of epithelioma of ear, Otol. 16
—— —— on deafness resulting from epidemic cerebrospinal meningitis, Otol. 87
—— —— on functional aphonia, Laryng. 80
—— —— on new adenotome, Laryng. 81
—— —— on operative surgery of labyrinthitis, Otol. 76
—— —— on sudden deafness in case of syphilis, Otol. 8
——, purulent otitis media with deep ulceration of external meatus in a case of secondary syphilis, Otol. 88; discussion (p. 88): Dr. Paterson, Dr. Grant
Bronzing, cutaneous, presence in cystic tumour of suprarenal body, rare, Surg. 215
Brown, T. E. Burton, C.I.E.—Discussion on action of digitalis on human heart, Therap. 31
—— discussion on arteriosclerosis, Therap. 37
——, presidential address at opening meeting of Therapeutical and Pharmacological Section, Therap. 1
Brown, W. Langdon.—Discussion on treatment of pneumonia, Med. 47
——, statistics of complications of pneumonia from St. Bartholomew's Hospital, Med. 55
Bruce, W. Ironside.—Discussion on cauterization as an adjuvant to radiotherapy, Electr. 90
—— discussion on electrolytic method of measuring X-rays, Electr. 100
—— —— on ionic medication, Electr. 77
—— —— on X-ray diagnosis of renal and ureteral calculi, Electr. 31
——, the treatment of leukæmia, exophthalmic goitre, sarcoma, &c., by X-rays, Electr. 55; discussion (p. 60): Dr. R. Morton, Mr. A. Edmunds, Dr. Donald Baynes, Dr. H. Lewis Jones, Dr. Hinds Howell, Dr. W. Ironside Bruce (reply)
—— and Wallis, F. C.—The treatment of ulcerative proctitis by zinc kataphoresis, Surg. 176; discussion (p. 184): Mr. Warrington Haward, Dr. Curtis Webb, Dr. H. K. Ramsden, Mr. Lockhart Mummery, Mr. Swinford Edwards, Mr. Wallis (reply), Dr. Ironside Bruce (reply)
Buchanan, G. S.—Discussion on medical inspection in schools, Epid. 69
Buckley, C. W.—Discussion on electrical treatment of atonic conditions of digestive system, Electr. 107
Bulbar paralysis, see *Paralysis* (bulbar)
Bullæ on face, case for diagnosis (Sir M. Morris), Derm. 17
Bulstrode, H. T.—Discussion on medical inspection in schools, Epid. 62, 68
Bunch, J. L.—Case of lichen spinulosus, Derm. 10
—— case of tuberculides in a girl, aged 11 years; Derm. 175; discussion (p. 175): Dr. C. Fox, Dr. G. Little
—— —— of tuberculosis developing on the site of a vaccination scar, Derm. 164
Burnet and Abrami, results of cutaneous tuberculin reaction, alluded to, Path. 75
Burns, J., views on menstruation, quoted, Obst. 292
Burt, A. Hamilton.—Case of septal deflection in a phthisical patient, Laryng. 111; discussion (p. 111): Dr. J. B. Ball, Mr. Barwell, Dr. Jobson Horne, Dr. Burt (reply)
Burt, J. Barnes.—Three cases of gout showing destructive changes in bone, Clin. 223; discussion (p. 230): Sir Dyce Duckworth
Busch, J. P. Zum.—Discussion on lymphangioplasty for brawny arm of breast cancer, Clin. 189
Bushnell, F. G.—Abscess of bone caused by an intermediate bacillus (P.) allied to *B. typhosus*, Path. 79
Butcher, W. Deane.—Discussion on cauterization as an adjuvant to radiotherapy, Electr. 88
——, discussion on case of gonorrhœal warts, Electr. 64
—— —— on diagnostic value of Röntgen rays in some diseases of chest, Electr. 44

Butcher, W. Deane.—Discussion on electrical treatment of atonic conditions of digestive system, Electr. 106
—— —— on electrolytic method of measuring X-rays, Electr. 99
—— —— on ionic medication, Electr. 76
——, plastic Röntgenography (exhibition of lantern slides), Electr. 64
——, the future of electricity in medicine (presidential address before Electro-Therapeutical Section), Electr. 1
Butler, W.—Discussion on etiology and epidemiology of typhoid fever, Epid. 225
——, discussion on medical inspection in schools, Epid. 66
—— —— on rubella, Epid. 129
Butyric acid, production of acid-fast quality in bacilli by, Otol. 105, 106
Buzzard, E. Farquhar.—Case of cervical caries, Neur. 69
—— case of disease of the cervical vertebræ, with pressure symptoms, Neur. 12; discussion (pp. 13, 14): Dr. Head, Dr. Wilson, Mr. Donald Armour, Dr. Grainger Stewart, Dr. James Taylor, Dr. Buzzard (reply)
—— case of Huntington's chorea, Neur. 12
—— —— of tubercle of one sixth nucleus, Neur. 38; discussion (p. 40): Sir Victor Horsley
—— —— of unusual vertebral deformity with seventh cervical ribs, Neur. 69
——, discussion on arteritis obliterans of right upper extremity associated with malformation of both clavicles, Neur. 102
—— —— on case for diagnosis, Neur. 94
—— —— on case of extreme lordosis of two years' standing in a woman aged 32, Neur. 59
—— —— on cerebral gummata, Neur. 20
—— —— on cervical caries, Neur. 65
—— —— on intracranial tumour, Neur. 104
—— —— on pathology of epilepsy, Med. 117
—— —— on "tics," Neur. 46
——, note on the occurrence of muscle-spindles in ocular muscles, Neur. 83; discussion (p. 87): Dr. F. E. Batten, Dr. Buzzard (reply)
Buzzard, Thomas.—Discussion on neuritis of brachial plexus, Clin. 32

Cabal, R. C., his recorded cases of lymphocythæmia mentioned, Clin. 132
Cæsarean hysterectomy for traumatic atresia of vagina (A. Routh), Obst. 1
—— —— for stenosis of vagina (K. Das), Obst. 188
Caiger, F. F.—Discussion on rubella, Epid. 126
Calcareous patches in atheromatous vessel causing confusion in X-ray diagnosis of calculi, Electr. 28
Calcium, see *Blood-calcium*
—— salts, deposition around straightened elastic fibres of vessels in arteriosclerosis, Therap. 34, 35
Calculi (vesical), action upon photographic plate in darkness (H. A. Colwell), Path. 127
Calculus (renal), diagnosis by X-rays (G. H. Orton), Electr. 15
—— —— —— —— conditions leading to confusion in diagnosis, Electr. 26
—— (ureteral), diagnosis by X-rays (G. H. Orton), Electr. 15
—— —— —— —— conditions leading to confusion in diagnosis, Electr. 26
Caley, H. A.—Discussion on complications of pneumonia, Med. 37
—— discussion on tone of cardiac muscle, Med. 272
—— and Langmead, F. S.—Statistics of complications of pneumonia from St. Mary's Hospital, 1896-1905, Med. 69
Callus, excessive formation of, diagnosed as sarcomatous growth, Path. 92
——, hypertrophied, of tibia and fibula, Path. 101
—— —— —— ——, diagnosis from rickets and congenital syphilis, Path. 104
—— —— —— ——, how differing from adult specimen of non-calcifying plastic osteitis of tibia, Path. 103
——, swelling of, formed after fractures, Path. 109
——, see also *Redundant callus formation*, Path. 107

Calmette, A., on the tuberculin ophthalmic reaction, Path. 75
Calomel, probable value as a parasiticide, Therap. 24
CAMERON, H. C.—Discussion on action of digitalis on human heart. Therap. 31
——, discussion on treatment of functional dyspepsia, Therap. 10
——, FAWCETT, JOHN, and MANN, H. C. C.—Statistics of complications of pneumonia from Guy's Hospital (1902-6), Med. 69
CAMERON, JOHN, on the pelvic fascia, quoted, Obst. 52, 53
—— on the supports of the pelvic viscera, quoted, Obst. 54
CAMPBELL, HARRY.—The therapeutics of diet (an address given at the Annual Conversazione of the Therapeutical Section), Therap. 45
Camphor, monobromide of, in treatment of acute stage of chorea, Therap. 76, 77
Cancer invading myoma, specimen of (H. Macnaughton Jones), Obst. 155
—— —— ——, report of Pathology Committee on, Obst. 156
—— of cervix, advanced, treatment by Wertheim's method of hysterectomy, Obst. 100
—— —— in woman, aged 26, treated by high amputation with cautery (H. R. Spencer), Obst. 31
—— (primary cutaneous) of chest involving neighbouring nerve areas (D. Walsh), Derm. 187
—— of larynx, treated by laryngectomy (C. J. Symonds), Clin. 184
—— of ovary, primary bilateral (T. W. Eden), Obst. 243
—— of right lobe of thyroid gland (J. Donelan), Laryng. 46
—— of stomach, treatment by Röntgen rays, Electr. 120
—— of uterus, perithelioma of ovary coexisting with (A. H. N. Lewers), Obst. 156
—— ——, removal of uterus and glands by Wertheim's method (C. H. Roberts), Obst. 61
—— ——, sarcomatous ovarian tumour coexisting with (A. H. N. Lewers), Obst. 69
—— (caseating) of body of uterus (H. R. Spencer), Obst. 119
——, see also *Epithelioma : Malignant* disease ; *Malignant* growth
Cannabis indica and physostigma in treatment of acute stage of chorea, Therap. 77
CANTLIE, JAMES.—Some tropical diseases and the remedies required for their treatment and prophylaxis, Therap. 22 ; discussion (p. 27) : Professor Cushny, Mr. Maben, Dr. J. Gray Duncanson, Mr. Cantlie (reply)
Captivity, tuberculosis most rife in birds kept in, Path. 22
Carbohydrates (fermentable), cause of dental caries, Odont. 34, 35, 36, 69
Carboluria and ochronosis, Clin. 58
Carcinoma and sarcoma, tumours consisting of, explanation, Path. 170
——, see also *Cancer*
Carcinomata, Malpighian or basal squamous cell, Path. 168
Cardiac arrest, evidence of, in idiopathic epilepsy, Med. 76
—— inhibition, causation of, in epilepsy, Med. 82
Carditis, rheumatic, treatment, Therap. 70, 85
—— ——, treatment of pain in, Therap. 71, 85
CARGILL, L. V., TURNER, W., and THOMSON, STCLAIR.—Case of abscess in left cerebral frontal lobe, originating from suppuration in the left frontal sinus, Laryng. 125 ; discussion (p. 125) : Dr. S. Spicer, Mr. Stuart-Low, Mr. H. Tilley, Mr. W. Turner (reply)
Caries (cervical), case of (E. F. Buzzard), Neur. 69
—— ——, operation and recovery (D. Armour), Neur. 64
—— (dental), etiology of, Odont. 34
—— —— ——, bread made from roller-milled white flour, Odont. 71, 72
—— —— ——, consumption of sweetstuffs, Odont. 35, 70, 71
—— —— ——, fermentable carbohydrates, Odont. 34, 35, 36, 69
—— —— ——, foodstuffs, Odont. 73
—— —— ——, resolution to appoint subcommittee of Odontological Section to consider and report on best method of arriving at knowledge of, Odont. 73, 78
—— ——, immunity to, due to reversion to type, Odont. 116
—— ——, influence of food in production of, Odont. 114, 123
—— ——, prevalence among poorer classes, Odont. 71, 72

Caries (dental), prevalence due to removal of influence of natural selection, Odont. 75, 111-117
—— ——, prevention of, means for, Odont. 37
—— (spinal), treatment of, appliance for obtaining extension of spine in (T. H. Openshaw, C.M.G.), Clin. 196
—— —— with clubbing of fingers and toes (P. M. Heath), Clin. 231
CARLESS, ALBERT.—Multiple symmetrical lipomata, Clin. 233
Carotid artery, see *Artery*
Carpus, fracture in, diagnosis by X-rays (A. D. Reid), Electr. 62
Carriers, chronic, of *Bacillus typhosus*, case in neighbourhood of New York, Epid. 211
—— —— —— ——, cure of condition, Epid. 188
—— —— —— ——, fallacy respecting infection by, Epid. 219
—— —— —— —— in asylums, Epid. 212, 213
—— —— —— ——, mechanism of infection by, Epid. 187
—— —— —— ——, more common among women than men, Epid. 177, 212
—— —— —— ——, prophylaxis against infection by, Epid. 188
—— —— —— ——, quiescent periods in infectiveness, Epid. 188
—— —— —— ——, spread of typhoid fever by, Epid. 175-191, 225, 226, 227
—— —— —— —— —— —— —— by same carrier at Brentry Certified Inebriate Reformatory, Bristol, Epid. 177
—— —— —— —— —— —— —— by same carrier at Grove House Home for Girls, Brislington, Epid. 185
CARRUTHERS, S. W.—Purpura hæmorrhagica with fatal result from cerebral hæmorrhage, Clin. 120; discussion (p. 122): Sir T. Barlow, Dr. J. Porter Parkinson, Dr. Carruthers (reply)
CARTER, JAMES T.—Some notes on the growth of the jaws, Odont. 17; discussion (p. 24): Mr. F. J. Bennett, Mr. N. G. Bennett, Mr. H. Baldwin, Mr. J. F. Colyer, Mr. Carter (reply)
CARVER, NORMAN C., and FAIRBAIRN, JOHN S.—Hæmorrhage into the pons Varolii as the immediate cause of death in the eclampsia of pregnancy, with illustrative cases, Obst. 79; discussion (p. 92): Dr. Herbert Spencer, Dr. Macnaughton Jones, Mr. A. Lionel Smith, Dr. Amand Routh, Mr. D. C. Rayner, Dr. R. H. Hodgson, Dr. Fairbairn (reply)
Case's classification of dento-facial irregularities, Odont. 141
Cataphoresis, see *Ionic medication*
Catarrh, acute suffocative, of Laennec (S. West), Med. 151
—— —— —— ——, conditions closely allied to, Med. 153, 156-158
—— —— —— —— ——, conditions from which it must be diagnosed, Med. 153-156
Cathcart, tabular analyses of excretion under starvation diet, quoted, Path. 139, 142
Cauterization, study of method of action enjoined, Electr. 89
Cautery, high amputation by means of, in case of cancer of cervix in woman aged 26 (H. R. Spencer), Obst. 31
Cells, enormous alteration in appearance may be caused by purely physical conditions, Path. 170
——, see also *Epithelial cells*
—— —— *Mesoblastic cells, Prickle cells*
Cellulitis, extensive, of right side of neck (P. Abercrombie), Laryng. 10
—— (pelvic), induction of, in cure of prolapse of uterus, Obst. 46
Cerebellum (abscess of), crossed abducens paralysis in (D. R. Paterson), Otol. 88
—— —— ——, nystagmus in, Otol. 136, 139, 142
—— —— ——, unusual case (D. R. Paterson), Otol. 23
—— —— ——, with phlebitis of lateral sinus in case of middle ear suppuration (P. McBride), Otol. 135
——, atrophy of (F. E. Batten), Clin. 18
——, tumour of (fibro-psammoma), with proptosis (J. P. Parkinson and J. S. Hosford), Clin. 121
——, see also *Ataxy* (cerebellar)

Cerebral disturbance of high order denoted by movements in chorea, Therap. 73
—— hæmorrhage, fatal, in case of purpura hæmorrhagica (S. W. Carruthers), Clin. 120
—— intravascular clotting as cause of epilepsy, Med. 93
Cerebrospinal fluid in diagnosis of meningitis, Otol. 141, 142
—— ——, presence of antibodies in, Surg. 160, 162
—— ——, removal of, by lumbar puncture in relief of uræmic symptoms, Med. 103
Cerein, suggested to be responsible for acid-fast property of aural bacteria, Otol. 106, 119
Cerium, oxalate of, combined with pepsin useful in dyspepsia, Therap. 17, 18
Cervicitis, treatment by ionic medication, Electr. 134
Cervix uteri, cancer of, in woman aged 26 treated by high amputation with autery (H. R. Spencer), Obst. 31
—— —— ——, advanced, treatment by Wertheim's method of hysterectomy (A. H. N. Lewers), Obst. 100
—— ——, ectropion of mucosa of, in infant (R. Jamison), Obst. 323
—— —— , fibroid of, complicating pregnancy; subtotal hysterectomy at fourth month (J. Bland-Sutton), Obst. 17
—— ——, sarcoma of, specimen (Miss L. G. Anderson), Obst. 153
—— —— —— ——, report of Pathological Committee on, Obst. 155
—— —— —— ——, grape-like, removal, with vaginal hysterectomy (F. J. McCann), Obst. 253
—— ——, tubercle of (P. Horrocks), Obst. 66
Chalk, wells in, potential danger of typhoid infection by water derived from, Epid. 191, 224
—— —— —— —— —— —— —— ——, experiments to prove, Epid. 192-200, 224
CHALMERS, Dr.—Discussion on plague, Epid. 97
CHAMPNEYS, F. H.—Discussion on abdominal hysterotomy for chronic uterine inversion, Obst. 288
—— discussion on case of difficult labour, Obst. 148
—— —— on myomectomy during pregnancy, Obst. 135
Characters, acquired, inheritance of, Odont. 110
——, hereditary, alternative pairs, Epid. 144
—— —— —— ——, existence in, essential feature of Mendel's discovery, Epid. 136, 139
—— —— —— ——, nature of, Epid. 146
—— —— —— —— ——, illustrated by crossing of Breda fowl with comb-bearing fowl, Epid. 148
—— —— —— ——, presence and absence hypothesis of relation between factors of, Epid. 148
—— ——, diversity of, showing Mendelian inheritance, Epid. 139
—— ——, dominant and recessive, Epid. 138
—— ——, gametic coupling of, Epid. 151
—— ——, interaction of, illustrated by crossing of sweet pea, Epid. 149
—— —— interdependence of, illustrated by crossing of rabbits, Epid. 145
—— ——, partial coupling between, Epid. 153
—— ——, segregation of factors representing, Epid. 136
Charcoal, transference of, through intact intestinal mucosa of rabbit, Path. 42
——, see also *Vegetable* charcoal
Charcot, hypnotic theories of, identical with those of Elliotson and other later mesmerists, Therap. 55
Charing Cross Hospital, statistics of complications of pneumonia at (W. Cecil Bosanquet), Med. 71
Charles, J. R., on effects of fright on children, quoted, Med. 92
Chasmorhynchus, aspergillosis in, Path. 20
Chavane and Maygrier, their case of cerebral hæmorrhage in eclampsia of pregnancy described, Obst. 86
Cheadle, W. B., on rubella, quoted, Epid. 118
CHEATLE, ARTHUR.—Discussion on case for diagnosis, Otol. 27
——, discussion on case of cerebellar abscess, Otol. 23
—— —— —— of deaf mute apparently regaining hearing, Otol. 13

Cheatle, Arthur.—Discussion on case of lateral sinus disease, Otol. 125
——, discussion on case of Ménière's disease, Otol. 26
—— —— —— of primary epithelioma of tympanum, Otol. 35
—— —— —— of stenosis of right auditory meatus, Otol. 11
—— —— on sudden deafness in case of syphilis, Otol. 9
—— —— on histological preparations of human cochlea, Otol. 19
——, nine specimens of fracture through the temporal bone, Otol. 10; discussion (p. 11): Dr. P. McBride, Mr. L. B. Rawling, Mr. Sydney Scott, Dr. Milligan, Mr. Cheatle (reply)
——, right temporal bone of a man aged 42, showing how pus may reach the neck without passing through the mastoid process, Otol. 24
Cheek, telangiectasis of (H. G. Adamson), Derm. 44
Cheinisse, on "fourth disease" quoted, Epid. 116
Chest, diseases of; diagnostic value of Röntgen rays in certain diseases of chest (A. Stanley Green), Electr. 35
——, primary cutaneous carcinoma of, involving neighbouring nerve areas (D. Walsh), Derm. 187
——, see also *Thorax*
Chicken-pox, changes in age- and sex-incidence before and during school life, Epid. 59
Childhood, rheumatism and chorea in (L. G. Guthrie), Therap. 65
Children, complications of pneumonia in, Med. 32, 33
——, dental caries in, etiology, Odont. 34
—— —— ——, prevention, Odont. 37
——, dental disease in, treatment (J. F. Colyer), Odont. 33
——, effects of fright on, Med. 92
——, favus and ringworm in, treatment by Röntgen rays prejudicial, Electr. 119, 120
Chill as an agent in causation of chorea compared with fright and shock, Therap. 72
——, motor insufficiency of stomach due to, Therap. 11
Chimpanzee, existence of intermaxillary antrum in, Odont. 22
China, South, see *Yunnan*
Chisel, preference for, in operative surgery of labyrinthitis, Otol. 81
Chloral in treatment of acute stage of chorea, Therap. 76, 81
Chloretine in treatment of acute stage of chorea, Therap. 77
Chloroform, anæsthesia by, in operation for goitre, Surg. 31
—— —— —— —— —— —— —— must be superficial, Surg. 31, 77
—— produces dilatation of heart, Med. 271, 272
——, value of, in irritable dyspepsia, Therap. 19, 20
Chlorosis, chronic constipation due to, Med. 140
Cholecystectomy, cases of (J. D. Malcolm), Surg. 93
——, question of performance, Surg. 97-105
——, recurrence of gall-stones after, rare, Surg. 97, 98, 106
——, risks of, Surg. 98, 100, 101, 102, 105
—— ——, hæmorrhage, Surg. 102, 103
Cholecystotomy, when to be preferred to cholecystectomy, Surg. 105, 106
Cholesteatoma, accompanying labyrinthitis, Otol. 57, 61, 63, 64, 66, 76
——, presence in aural discharges, Otol. 114
——, presence in antro-tympanic cavity indicated by acid-fast property of old non-nucleated squames in aural discharge, Otol. 99, 121
Chondroma, osteoid, case of, Path. 110
—— —— ——, histological description, Path. 111
Chorea, age-incidence, Therap. 74, 82
——, analysis of cases, Therap. 73
——, cardiac affection in, percentage, Therap. 74
——, changes in age- and sex-incidence before and during school life, Epid. 58
——, duration of attacks, Therap. 74

Chorea, etiology of, Therap. 72
—— ——, paramount factors, Therap. 72
—— in childhood (L. G. Guthrie), Therap. 65
——, mortality of, Therap. 78, 87
——, movements of, differ from those of tics, Neur. 43
—— ——, imply cerebral disturbance of high order, Therap. 73
——, occasional development of tics from, Neur. 46
——, psychomotor manifestation of rheumatism, Therap. 73
——, relapsing cases, Therap. 74
——, relation to rheumatism, Therap. 71
—— —— —— shown by history of cases, Therap. 74
——, seasonal incidence of, Therap. 74
——, treatment, Therap. 75
—— —— of acute stage, Therap. 76
—— —— in residual stage, Therap. 76
——, see also *Huntington's* chorea
—— mollis, treatment, Therap. 77
Chorionepithelioma of testicle, histological discovery of, Path. 1
—— of uterus with extensive vaginal and visceral metastases (T. W. Eden), Obst. 13
Choroid plexus of fourth ventricle, tumour of, Neur. 28, 30
CHURCH, Sir WILLIAM SELBY, Bt., K.C.B.—Discussion on growth of malignant disease, Surg. 18
——, his case of pneumonia complicated by endocarditis referred to, Clin. 118
Cibiculture, era of, proportion of vegetable to animal food increased under, Therap. 46, 47
Circulatory changes, correlation with symptoms and recovery from epileptic attack, Med. 94
Cirrhosis of suprarenal capsules found post mortem in case of leucodermia with symptoms of Addison's disease (N. Dalton), Clin. 209
Clarke, F. H., on results of stimulation of peripheral end of vagus, Med. 84
Clarke, Sir G. S., on value of plague inoculation, quoted, Epid. 80
Claudication (intermittent) in lower extremity (angina pedis or angina cruris) from chronic arteritis obliterans (F. Parkes Weber), Clin. 44, Neur. 50
Clavicles (both), malformation of, in association with arteritis obliterans of right upper extremity (L. G. Guthrie), Neur. 101
Clinical Society of London, record of work of (Sir T. Barlow, presidential address), Clin. 1
Clockwork resistances, control of interrupted currents by, Electr. 115
—— ——, see also *Metronome*
Clonic spasms, cause of, Med. 98
Clostridia in aural discharges, Otol. 105
Clothing, defects of, in school children, Epid. 53
—— source of typhoid infection, Epid. 222
Cocaine, application previous to zinc cataphoresis, Surg. 184
—— ions in treatment of neuralgia, Electr. 76
Coccygeus muscle, anatomy and functions, Obst. 208
Cochlea, anatomy, Otol. 46
——, histological preparations (S. R. Scott), Otol. 19
——, partial removal of, with double vestibulotomy in labyrinthitis, Otol. 64
COCKE, R. S.—Case for diagnosis, Otol. 27; discussion (p. 27): Dr. McBride, Dr. Jobson Horne, Mr. Cheatle, Dr. Pritchard, Mr. West, Mr. Waggett, Mr. Fagge, Mr. Whitehead, Mr. Kelson, Dr. W. Hill, Mr. Scott
CODD, J. A.—Discussion on diagnostic value of Röntgen rays in some diseases of the chest, Electr. 53
COHEN, Dr., and GALLOWAY, JAMES.—Case of acute scarlatiniform eruption following the administration of small doses of quinine, Derm. 71
Colectomy, doubtful benefit of, in chronic constipation, Med. 125
COLEMAN, F.—Discussion on case of extensive necrosis of mandible, Odont. 31

Colitis, treatment by electric enemata, Surg. 177
—— (croupous) complicating pneumonia, Med. 13
—— (follicular ulcerative), treatment by zinc cataphoresis, Surg. 177
—— (ulcerative acute) fatal case with unusual symptoms (S. Phillips), Med. 161
—— —— ——, question of isolation of bacillus, Med. 163, 164, 165, 167
—— —— —— ——, of preceding disease, Med. 165
—— —— ——, relation to dysentery, Med. 163-167
—— —— ——, resemblance to kala-azar, Med. 165
—— —— ——, treatment by operation advocated, Med. 165, 166
—— (ulcerative, post-operative), treatment by zinc cataphoresis, Surg. 177
Collateral fluxion, relation to suffocative catarrh, Med. 153, 157
Collier, J. S.—Discussion on cases of myatonia congenita and of toxic affection of muscles and lower neurones, Neur. 3
——, discussion on "tics," Neur. 45
——, some peculiarities of cerebral gummata, Neur. 15; discussion (p. 19): Sir Victor Horsley, Dr. Purves Stewart, Dr. Farquhar Buzzard, Dr. Collier (reply)
——, three cases of amyotonia congenita, Neur. 60; discussion (p. 61): Dr. F. E. Batten, Dr. Head, Dr. C. E. Beevor (reply)
Collins, E. Treacher, and MacLeod, J. M. H.—Two cases of advanced "keratosis follicularis associated with baldness," Derm. 27
Collum valgum, see *Coxa valga*
Colotomy in treatment of ulcerative proctitis, Surg. 180, 181, 182
Colon (Isthmus of Panama), insanitation, till recently, notorious, Epid. 306, 307
—— provided with water supply, Epid. 309
Colour-blindness, inheritance of, Epid. 157
Colpotomy (posterior), with incision of entire uterus, in treatment of chronic uterine inversion, Obst. 283, 286
Colwell, Hector A.—On the action of vesical calculi upon a photographic plate in the dark, Path. 127
Colyer, J. F.—Discussion on classification of dento-facial irregularities, Odont. 145
—— —— on diffuse fibromata of gums, Odont. 128
—— —— on growth of jaws, Odont. 25
—— —— on incomplete eruption of temporary molars, Odont. 100
——, exhibition of specimens added to Museum of Odontological Section, Odont. 27
——, the treatment of children from the dental aspect, Odont. 33; discussion (p. 40): Mr. Howard Mummery (adjournment)
—— —— —— —— —— discussion (pp. 68-78): Mr. William Hern, Mr. C. Robbins, Mr. C. N. Peacock, Mr. W. Rushton, Mr. F. J. Bennett, Mr. Martin Henry, Dr. Sim Wallace, Mr. Stanley Mummery, Mr. Stanley Colyer, Mr. Sturridge, Mr. Baly, Mr. J. F. Colyer (reply)
Colyer, Stanley.—Discussion on treatment of children from dental aspect, Odont. 76
Coma, post-epileptic, and sleep, Med. 99
——, uræmic, Med. 104
Comedones (grouped), case in infant (J. M. H. MacLeod), Derm. 14
Conjunctivæ, injection of, in rubella, Epid. 103
Conjunctivitis (phlyctenular) and ulceration of eyes in association with well-marked rosacea (E. G. Graham Little), Derm. 181
Connective tissues, visceral, dependent upon efficiency of pelvic floor for integrity, Obst. 204
—— —— ——, non-elasticity of, Obst. 203
—— —— ——, support of pelvic viscera, Obst. 195, 204
Conradi, views on regional immunity from typhoid fever quoted, Epid. 206-209
—— on typhoid "carriers" quoted, Epid. 210
Constipation at onset of acute ulcerative colitis, Med. 161, 163
——, chronic (A. F. Hertz), Med. 119-148
—— —— due to chlorosis, Med. 140

Constipation, chronic, due to lead poisoning, Med. 137
—— —— due to tabes dorsalis, Med. 135
—— ——, examination of cases under X-rays, Med. 120-147
—— ——, history of cases, Med. 122-148
—— —— of unknown origin, Med. 144
—— ——, treatment (A. F. Hertz), Med. 119-148
—— —— —— by daily use of enema, Med. 126
—— —— —— by electricity, Electr. 101, 108, 109
—— —— —— by operation, of doubtful benefit, Med. 125, 132
—— ——, with enteroptosis, Med. 142
—— ——, with neurasthenia, Neur. 147
Convulsions accompanying pulmonary embolism, Med. 97
——, epileptic, cause of, Med. 96
——, infantile, connection with epilepsy, Med. 91
——, Jacksonian, Med. 99
——, uræmic, Med. 105
Cookery, beginnings of, among human race, Therap. 46
Coördinated movements as test for presence of labyrinthitis, Otol. 70
Copeman, S. M.—Discussion on lymphangioplasty for brawny arm of breast cancer, Clin. 189
Coplans, Myer.—Medical inspection in schools: the Gloucestershire scheme, Epid. 45; discussion (pp. 62-70): Dr. Bulstrode, Dr. J. M. Martin (communication by letter), Dr. Sandilands, Dr. H. Meredith Richards, Lieut.-Colonel Macpherson, R.A.M.C., Dr. Butler, Dr. Hamer, Dr. Buchanan, Dr. Coplans (reply)
Copper ions in treatment of diphtheritic paronychia, Electr. 74
—— —— —— of lupus erythematosus, Electr. 80
—— —— —— of ulceration of mouth, Electr. 75
Corbin, H. E.—Rubella: its identity and pathology, Epid. 117; discussion (p. 124): Sir S. Murphy, Dr. Ransome, Dr. Caiger, Dr. Meredith Richards, Dr. F. N. Hume, Dr. Butler, Dr. Claude B. Ker, Dr. J. T. C. Nash, Dr. Corbin (reply)
Corn Laws, repeal of, effects in Ireland, Epid. 14
Cornea, ulceration of, treatment by zinc ions, Electr. 75
Corpus luteum, relation of ovum to, in specimens of ovarian associated with intra-uterine pregnancy, Obst. 275
Courtauld, L., and Leitch, A.—The occurrence of lumina in malignant tumours, Path. 173
Coxa valga (A. H. Tubby), Surg. 107-140
—— ——, abduction and rotation out of limb in, Surg. 136
—— ——, angle of inclination in, Surg. 109, 110
—— ——, compensatory changes in trunk in, Surg. 136
—— ——, diagnosis, Surg. 137
—— ——, flattening of region over great trochanter in, Surg. 136
—— ——, gait in, Surg. 135
—— ——, importance of careful skiagraphy in, Surg. 136
—— ——, lengthening of limb in, Surg. 135
—— ——, museum specimens of, Surg. 110, 115
—— ——, pain and spasm in, Surg. 135
—— ——, pathogenesis, Surg. 134
—— ——, posture in, Surg. 136
—— ——, relative frequency, Surg. 113
—— ——, symptoms and signs, Surg. 135, 136
—— ——, treatment, Surg. 137
—— —— —— by linear osteotomy, Surg. 138
—— —— (acquired) association with infantile paralysis, Surg. 125
—— —— ——, association with rickets, Surg. 133
—— —— —— due to traction exerted by pendent limb, Surg. 125

Coxa valga, acquired, following forceps delivery, Surg. 112
—— —— —— —— fracture and malunion of shaft of femur, Surg. 129
—— —— —— from rickets, Surg. 113
—— —— —— from separation of epiphysis, Surg. 112
—— —— ——, idiopathic, Surg. 133
—— —— ——, static or functional varieties, Surg. 128
—— —— ——, traumatic, Surg. 111, 118, 133
—— —— (congenital) not associated with other abnormalities, Surg. 121
—— —— ——, with congenital dislocation of hip, Surg. 121
Crab, see *Land crab*
Craniotomy, value of version as means of delivery after, Obst. 148
Cranium, growth of, influenced by growth of jaws, Odont. 23
Creatinin, excretion of, in lying-in women (C. N. Longridge), Obst. 193
Cricoid cartilage, malignant growth behind (J. W. Bond), Laryng. 111
CRISP, E. A.—Case for diagnosis (shown by Dr. J. J. Pringle), Derm. 18
CROCKER, H. RADCLIFFE.—Case illustrating the effect of X-rays on mycosis fungoides, Derm. 75
——, case of mycosis fungoides, Derm. 167; discussion (p. 168): Dr. Pernet, Mr. Hartigan (reply)
——, discussion on case for diagnosis (factitious ulcers), Derm. 70
—— —— —— —— —— (feigned herpetiform eruption), Derm. 83
—— —— —— —— —— (granuloma of mouth with penile ulcer), Derm. 58
—— —— —— —— —— (? leprosy), Derm. 53
—— —— case of acute lichen planus, Derm. 33
—— —— —— of bilateral telangiectasis of trunk, Derm. 69
—— —— —— of bromide eruption, Derm. 75
—— —— —— of chronic X-ray dermatitis of hands, Derm. 80
—— —— —— of (?) dermatitis artefacta, Derm. 85
—— —— —— of disseminated lupus, Derm. 94
—— —— —— of folliculitis decalvans (pseudo-pelade of Brocq), Derm. 86
—— —— —— of lichen spinulosus, Derm. 51
—— —— —— of lupus with unusual features, Derm. 78
—— —— cases of lupus erythematosus, Derm. 46, 171
—— —— case of lupus erythematosus, saccharomycetiform, Derm. 94
—— —— —— of macular atrophy of scalp, Derm. 60
—— —— —— of multiple leiomyoma, Derm. 50
—— —— —— of nodular ringed eruption, Derm. 82
—— —— —— of ringed eruption, Derm. 48
—— —— —— of ringed eruption on hand, Derm. 65
—— —— —— of rodent ulcer, Derm. 62
—— —— —— of scarlatiniform eruption following administration of quinine, Derm. 73
—— —— —— of telangiectasis of cheek, Derm. 45
—— —— —— of varus nodulosus of Brooke, Derm. 85
—— —— —— of xanthoma of diabetic type, Derm. 93
—— —— cases of elephantiasis græcorum, Derm. 56
—— —— on granuloma annulare, Derm. 160
—— —— on sclerodermia (morphœa), Derm. 163
—— —— on specimens from case of syphilis, Derm. 174
——, exhibition of drawing of case for diagnosis, Derm. 91
——, presidential address before Dermatological Section, Derm. 1
—— and PERNET, GEORGE.—Case for diagnosis, Derm. 36; discussion (p. 38): Dr. J. M. H. MacLeod, Dr. Pringle, Dr. Leslie Roberts
—— ——, case of lichen planus verrucosus treated by violet light, Derm. 38
—— ——, senile warts developing into fungating growths, Derm. 56
—— ——, two cases of elephantiasis græcorum, Derm. 53; discussion (p. 56): Dr. Colcott Fox, Dr. Whitfield, Dr. Radcliffe Crocker, Dr. Rost, Dr. Pringle, Dr. Pernet (reply)

Crocker, H. Radcliffe, and Pernet, George.—Unusual case of pustular vegetating dermatitis, with pigmentation changes, in a woman aged 26, Derm. 168; discussion (p. 168): Mr. Hartigan, Dr. Pernet (reply)

Crossoptilon (Mantchurian), parasitic invasion of, simulating tubercular infection, Path. 21

Crotchet, value of, in difficult labour, Obst. 149

Croup, changes in age- and sex-incidence before and during school life, Epid. 57

Cruro-scrotal region, intertriginous dermatitis of, microscopical specimen and pure culture of yeast derived from (A. Whitfield), Derm. 190

Culex mosquito, extra-corporeal life of parasite of filaria in, Therap. 24, 25

—— —— —— —— —— of parasite of malaria in, basis of mosquito-malaria theory, Therap. 24, 25

Cullingworth, Dr. Charles James, death of, Obst. 195

Cunningham, H. H. B.—Deafness resulting from epidemic cerebrospinal meningitis (two cases), Otol. 83; discussion (p. 86): Dr. P. McBride, Mr. Yearsley, Dr. Pritchard, Dr. Milligan, Dr. Gray, Dr. Bronner, Dr. Cunningham (reply)

——, discussion on histological preparations of the human labyrinth, Otol. 94

Curettage, simple, in labyrinthitis, Otol. 66

Currant-moth, see *Abraxas grossulariata*

Curtis, Henry.—Two cases of congenital absence of both thumbs, &c., Clin. 236

Cushing, H, on intracranial pain, alluded to, Med. 89

Cushny, Professor A. R.—Discussion on action of digitalis on human heart, Therap. 31

——, discussion on arteriosclerosis, Therap. 36

—— —— on tone of cardiac muscle, Med. 271

—— —— on tropical diseases and remedies, Therap. 27

——, nutmeg poisoning, Therap. 39; discussion (p. 42): Dr. F. B. Power, Dr. J. Gray Duncanson, Prof. Cushny (reply)

Cutaneous web (congenital), case of partial occlusion of both anterior nares (G. K. Grimmer), Laryng. 66

Cyanosis absent in case of enlarged spleen with polycythæmia (R. G. Hann), Clin. 59

—— in splenic polycythæmia (Prof. W. Osler, F.R.S.), Clin. 41

Cyst, pure, represented in cases of goitre removed by operation, Surg. 25

Cystic tumour of suprarenal body, cases recorded (A. H. G. Doran), Surg. 201-217

Cystoma or lipoma of neck (W. Jobson Horne), Laryng. 38

Cysts (secondary), implantation, connected with omentum in case of ovarian dermoid (M. Randall and T. W. P. Lawrence), Obst. 105, 109

—— —— —— —— —— —— —— ——, fate of, Obst. 116

——, see also under names of organs

Cytological examination of discharge in cases of middle ear and maxillary sinus suppuration (J. M. Darling), Otol. 163

Czermak, Johann Nepomuk (1828-1873), autograph letter by, exhibited (W. Jobson Horne), Laryng. 83

Dally, J. F. Halls.—Discussion on diagnostic value of Röntgen rays in some diseases of chest, Electr. 45

——, discussion on ionic medication, Electr. 78

Dalton, Norman.—Case of leucodermia which died with symptoms of Addison's disease, and in which cirrhosis of the suprarenals was found, Clin. 209; discussion (p. 212): Sir Thomas Barlow, Sir Dyce Duckworth, Dr. Parkes Weber, Dr. Dalton (reply)

——, discussion on acute ulcerative colitis, Med. 165

—— —— on case of hairball in stomach, Clin. 214

—— —— on complications of pneumonia, Med. 31

Danysz's rat virus, Epid. 76, 91

Darkness, action of vesical calculi on photographic plate in (H. A. Colwell), Path. 127

Darling, John M.—Preliminary note on cytological examination of the discharge in cases of middle ear and maxillary sinus suppuration, Otol. 163; discussion (p. 163): Dr. Logan Turner, Dr. Darling (reply)

Das, K.—Gastroschisis, Obst. 323

——, three cases of Cæsarean hysterectomy for stenosis of the vagina, Obst. 188; discussion (p. 192): Dr. H. Spencer, Dr. A. Routh

Dauber, John H.—Case of pregnancy complicated with large retroperitoneal and subligamentous fibromyoma, Obst. 132; discussion (p. 135): Dr. Herbert Spencer, Dr. Champneys, Dr. Herman, Dr. Macnaughton Jones, Dr. Amand Routh, Dr. Eden, Dr. Arthur Giles, Mrs. Boyd

Davenport, Kirk.—Discussion on treatment of pyorrhœa, Odont. 66

Davies, Col.—Discussion on etiology and epidemiology of typhoid fever, Epid. 224

Davies, D. S., and Hall, I. Walker.—Typhoid carriers, with an account of two institution outbreaks traced to the same "carrier," Epid. 175, 226

Davis, H. J.—A new adenotome, Laryng. 81; discussion (p. 81): Dr. Bronner, Dr. Grant

——, case of cyst on the floor of the nose, Laryng. 97; discussion (p. 97): Mr. H. B. Robinson, Dr. McKenzie, Mr. Clayton Fox

——, case of growth on the left vocal cord, Laryng. 96; discussion (p. 96): Dr. D. Grant

—— —— of nasal obstruction, Laryng. 75; discussion (p. 75): Dr. J. B. Ball, Dr. Grant, Dr. Smurthwaite, Dr. H. Tilley, Dr. Davis (reply)

——, discussion on case of functional aphonia in a male, Laryng. 78

—— —— —— of intranasal hypertrophy, Laryng. 47

—— —— —— of tumour of right thyroid lobe, Laryng. 39

—— —— —— of ulceration and destruction of epiglottis, Laryng. 34

—— —— —— of unilateral paralysis of tongue, Laryng. 54

—— —— on lipoma or cystoma of neck, Laryng. 38

—— —— on new instrument for passing suture through epiglottis, Laryng. 96

—— —— on preparations illustrating diseases of trachea, Laryng. 38

—— —— on subglottic laryngeal tumour, Laryng. 91

——, further notes on the case of a woman aged 27, with almost complete nasal obstruction on left side with inability to clear the nose, Laryng. 139; discussion (p. 140): Mr. Paterson

——, specimen from the case of a woman with a supposed cyst on the floor of the right naris, Laryng. 139

—— —— of a large pedunculated fibroma (? fibromyxoma) removed from the vault of the naso-pharynx of a boy aged 9, Laryng. 138

—— —— showing normal and hypertrophied pharyngeal tonsils in Southdown sheep, Laryng. 138

——, X-ray photograph showing a Hajek's hook in right sphenoidal sinus, Laryng. 138

Dawson, G. W.—Case for diagnosis, Derm. 25

——, case of lichen planus, Derm. 13

—— —— of pityriasis rubra pilaris, Derm. 13

—— —— of ringed eruption on the hand, Derm. 65

——, parakeratosis variegata, Derm. 175; discussion (p. 176): Dr. T. C. Fox

Deaf mute apparently regaining hearing (L. A. Lawrence), Otol. 13

Deafness in labyrinthitis, Otol. 56

—— resulting from epidemic cerebrospinal meningitis, two cases (H. H. B. Cunningham), Otol. 83

——, sudden, occurring under treatment for tertiary syphilis, effect of treatment upon (R. Lake), Otol. 7

——, see also *Nerve deafness*

Deciduoma malignum, histological discovery of, Path. 1; see also *Chorion-epithelioma*

Deformity, reason for removal of goitre, Surg. 23

Degeneration (cerebral) with changes at macula in four members of family (M. S. Mayou), Neur. 98

Delfino, case of macroglossia neurofibromatosa, quoted, Path. 9

Delivery, method of, after pubiotomy, Obst. 23

——, value of version as means of, after craniotomy, Obst. 148

Dental surgery, uses of paraform in (J. E. Spiller), Odont. 41
—— ——, see also *Teeth*
Dentine, presence of nerve fibres in, Odont. 47, 48, 49
—— —— —— —— —— ——, methods employed for demonstration of, Odont. 50-56
——, sensibility of, Odont. 45, 46, 47
Dento-facial irregularities, classification of, Angle's, Odont. 141, 145
—— —— —— —— —— ——, Case's, Odont. 141
—— —— —— —— ——, new method of (J. Sim Wallace), Odont. 140
—— —— ——, irregularities of individual teeth not related to, Odont. 142, 143
De-opsonization, strict specificity of, Med. 177, 179, 181
Dermatitis ? artefacta (E. G. Graham Little), Derm. 83
—— herpetiformis of unusual type (J. Galloway), Derm. 177
—— (intertriginous) of cruro-scrotal region, microscopical specimen and pure culture of yeast derived from (A. Whitfield), Derm. 190
—— papillaris capillitii (Kaposi) (J. M. H. MacLeod), Derm. 166
—— perstans, case for diagnosis (J. J. Pringle), Derm. 18
—— (pustular vegetating), unusual case, with pigmentation changes (H. R. Crocker and G. Pernet), Derm. 168
—— (seborrhoic), lichen spinulosus associated with (J. M. H. MacLeod), Derm. 51
——, X-ray (W. Fox), Derm. 177
—— —— (chronic) of hands, removal of warts by measured doses of X-rays (J. H. Sequeira), Derm. 78
Dermoid tumour (ovarian) retained two years in pelvis after obstructing labour (A. Doran), Obst. 35
—— ——, case with secondary cysts connected with omentum (M. Randall), Obst. 105
—— —— —— —— —— —— ——, extirpation, Obst. 106, 107, 117
—— —— —— —— —— —— ——, pathological report of case and of other cases recorded (T. W. P. Lawrence), Obst. 105, 108
—— —— —— —— —— —— ——, rupture of, cause, and results to peritoneum, Obst. 115
Derry on the pelvic fascia, quoted, Obst. 52
—— on supports of pelvic viscera, quoted, Obst. 196
De Santi, P. R. W.—Case of bilateral swelling of the septum nasi, Laryng. 93; discussion (p. 93): Dr. W. Williams, Dr. J. B. Ball, Mr. H. Tilley, Dr. Pegler, Mr. De Santi (reply)
——, discussion on atresia of naso-pharynx, Laryng. 16
—— —— on case of bilateral sarcoma of upper maxillæ, Laryng. 21
—— —— —— of extensive epithelioma of epiglottis, Laryng. 81
—— —— —— of intrinsic neoplasm of left vocal cord, Laryng. 90
—— —— —— of laryngeal disease, Laryng. 12
—— —— —— of multiple telangiectases, Laryng. 70
—— —— —— of necrosis of the arytænoid cartilage, Laryng. 65
—— —— —— of tumour of vocal cord, Laryng. 9
—— —— on extreme form of septal deflection, Laryng. 6
—— —— on extensive cellulitis of right side of neck, Laryng. 10
—— —— on fistulous opening in middle line of neck, Laryng. 3
—— —— on laryngeal disease, Laryng. 120
—— —— on malignant growth of septum and naso-pharynx removed by external osteoplastic operation, Laryng. 118
—— —— on new instrument for intralaryngeal operations, Laryng. 25
—— —— pulsating angeioma of the nose, Laryng. 142
—— —— tuberculosis and syphilis in larynx, Laryng. 137
Desiccation in cases in small-pox epidemic in Trinidad, Epid. 268
Desquamation and inflammation (chronic) of lips (J. M. H. MacLeod), Derm. 4
Diabetes, see *Xanthoma*, multiple, of diabetic type
—— insipidus, pedigree illustrating inheritance of, Epid. 143
Diagnosis, cases for (E. G. Graham Little), Derm. 164

Diagnosis, cases for (G. Ogilvie), Neur. 93, 94
—— ——, bullæ on face (Sir M. Morris), Derm. 17
—— ——, dermatitis perstans (J. J. Pringle), Derm. 18
—— ——, drawing exhibited (H. Radcliffe Crocker), Derm. 94
—— ——, eruption of extremities (G. W. Dawson), Derm. 25
—— ——, factitious ulcers (W. S. Fox), Derm. 70
—— ——, granuloma of mouth, with penile ulcer (J. H. Sequeira), Derm. 57
—— ——, herpetiform eruption (H. G. Adamson), Derm. 82
—— ——, keratosis (W. T. Freeman), Derm. 14
—— ——, laryngeal disease (F. Spicer), Laryng. 11; (R. H. Scanes Spicer), Laryng. 12
—— ——, (?) leprosy (Sir M. Morris for Dr. Kay), Derm. 52
—— ——, lymphangioma circumscriptum (E. G. Graham Little), Derm. 49
—— ——, swelling on mastoid process (R. S. Cocke), Otol. 27
—— ——, (?) xantho-erythrodermia perstans or parakeratosis variegata (H. Radcliffe Crocker and G. Pernet), Derm. 36-38
Diaphragm, movements of, measurement by orthodiagraph, Electr. 47
——, permanent paralysis of, resulting in case of acute anterior poliomyelitis (W. Pasteur), Clin. 51
——, pelvic, elasticity of, Obst. 202
—— ——, evolution of, Obst. 210
—— ——, failure of, as means of support to pelvic viscera, Obst. 204
—— ——, muscles of, Obst. 49, 200
Dickson, Dr., and Lassalle, Dr., account of origin of small-pox epidemic in Trinidad, 1902-4, quoted, Epid. 230
Diet, daily, in dyspepsia due to irritable stomach, Therap. 15
——, dry, value of, in treatment of gastric motor insufficiency, Therap. 7
——, human, evolution in, Therap. 45
—— —— —— ——, stages of, Therap. 45, 46
Dietetic neutralization of hyperacidity in gastric secretion, Therap. 5
Diets, ordinary and starvation, high coefficients of ammonia in urine under, Path. 138, 139
Digestion, psychical aspect of, Therap. 13
Digestive system, atonic conditions due to defective innervation, treatment by electricity, Electr. 101
—— —— —— —— due to true atony of muscular coat of digestive canal, treatment by electricity requires care and special method, Electr. 102-105
—— ——, disorders of, complicating pneumonia, Med. 40
—— ——, see also *Dyspepsia*
Digitalis, action on heart, Med. 258-261, 262, 270, 271, 272, 273, 274
—— —— —— (J. Mackenzie), Therap. 29
—— —— ——, beneficial in dilatation, 29
—— —— ——, effect of nervous system on, Med. 271
—— —— ——, effects on conductivity and contractility of cardiac muscle, Med. 264, Therap. 30
—— —— ——, inhibitory, Therap. 31
—— —— ——, irregularities, Med. 262
—— —— ——, little effect on rate under normal conditions, Therap. 29
—— —— ——, non-beneficial in cases of failure without dilatation, Therap. 30
—— —— ——, restores function of tonicity, Therap. 30
—— —— ——, slowing of pulse-rate in rheumatic cases, Therap. 30
—— in treatment of rheumatic carditis, how far efficacious, Therap. 70, 85
Dihybridism, Epid. 144
—— illustrated by crossing of peas, Epid. 144
Diphtheria, changes in age- and sex-incidence before and during school life, Epid. 57
——, dilatation of left ventricle in, Med. 273
—— of trachea, preparation illustrating (W. Jobson Horne), Laryng. 37
——, see also *Serum* (anti-diphtheritic)
Diphtheritic paronychia, treatment by copper ions, Electr. 74

Diplegia, cerebral (non-congenital), two cases of, in children (L. Guthrie), Neur. 67

Diplococcus catarrhalis in aural discharges, Otol. 108, 114

—— *intracellularis* (Weichselbaum's bacillus), presence in cases of cerebrospinal meningitis, Surg. 144, 165

—— *pneumoniæ* in aural discharges, Otol. 114

Disease, relation of poverty to, Epid. 2

—— —— —— ——, as illustrated by course of typhus fever and phthisis in Ireland (A. Newsholme), Epid. 1-44

Diseases, how far subject to laws of Mendelism, Epid. 160

——, sex-limited, Epid. 153

Disinfection of articles in prevention of spread of plague, difficulties attending, Epid. 74

Dislocation of left hip, traumatic, replaced by manipulation thirteen months later (T. H. Openshaw), Clin. 43

—— of humerus, dissection from behind, Surg. 190, 191

—— ——, dissection from, in front, Surg. 188, 189

—— ——, unreduced, Surg. 198

—— —— (subcoracoid, old), exposure from behind, Surg. 193

—— —— —— ——, reduction by excavating glenoid cavity through posterior intermuscular incision (W. G. Spencer), Surg. 187

—— —— —— —— —— —— —— —— —— —— —— ——, advantages, Surg. 197

—— of patella, ? congenital; operation, cure (D. Drew), Clin. 11

—— of shoulders (recurrent) cured by operation (T. H. Openshaw, C.M.G.), Clin. 29

Dixon, W. E.—Arteriosclerosis, Therap. 33; discussion (p. 36): Professor Cushny, Dr. T. D. Savill, Mr. Maben, Dr. Burton-Brown, Dr. Dixon (reply)

—— and Taylor, Frank E.—An epidiascopic demonstration on "The Physiological Action of the Placenta," Obst. 11; discussion (p. 11): Dr. H. Spencer, Dr. Macnaughton Jones, Dr. Amand Routh, Dr. W. S. A. Griffith, Dr. C. Nepean Longridge

Dog, effect of hypnotism on, Therap. 61, 62

—— suffering from ataxia and involuntary movements, pathological changes in nervous system of (F. E. Batten and G. Holmes), Neur. 22

Doganoff and Moro, cutaneous tuberculosis inoculated by v. Pirquet's method, Path. 75

Dohi, on staining for *Spirochæta pallida*, quoted, Obst. 168

Donelan, James.—Case of functional aphonia, Laryng. 79; discussion (p. 79): Dr. P. McBride, Dr. de H. Hall, Sir F. Semon, Mr. C. Horsford, Mr. M. Hovell, Dr. S. Spicer, Dr. Bronner, Dr. Smurthwaite, Dr. Donelan (reply)

——, case of infiltration of the vocal cord, Laryng. 83; discussion (p. 84): Mr. C. Fox

—— —— of intranasal hypertrophy with sweating of the nose, Laryng. 47; discussion (p. 47): Dr. J. B. Ball, Dr. Davis

—— —— of tumour of right thyroid lobe, Laryng. 39; discussion (p. 39), Dr. H. J. Davis, Sir Felix Semon, Dr. FitzGerald Powell, Dr. Ball, Dr. de Havilland Hall, Mr. Herbert Tilley, Dr. Dundas Grant, Dr. Donelan (reply)

——, discussion on case of girl aged 17, with a genuine baritone voice, Laryng. 61

—— —— on extreme form of septal deflection, Laryng. 6

—— —— on growth of middle turbinate body, Laryng. 2

—— —— on impaction of bone in larynx, Laryng. 5

—— —— on keloid following mastoid operation, Otol. 21

—— —— on lipoma or cystoma of neck, Laryng. 38

—— —— on neoplasm of right tonsil, Laryng. 141

—— —— on operative surgery of labyrinthitis, Otol. 80

—— —— on subglottic laryngeal tumour, Laryng. 90

——, specimen and microscopic section of a tumour of the right lobe of the thyroid gland, Laryng. 46; discussion (p. 47): Dr. J. B. Ball

——, two cases illustrating the effects of long-neglected adenoids on the development of the upper jaws and nasal septum, Laryng. 84; discussion (pp. 84, 85): Dr. J. B. Ball, Dr. S. Spicer, Dr. F. Powell, Mr. Herbert Tilley, Dr. F. H. Westmacott, Dr. L. H. Pegler, Mr. H. Barwell, Dr. Donelan (reply)

Donnan, views on causation of leucocythæmia, quoted, Clin. 133

Doran, Alban H. G.—Cystic tumour of the suprarenal body successfully removed by operation, with notes on cases previously published (with references), Surg. 201-217; discussion (p. 218): Mr. Lockwood, Mr. Alban Doran (reply)

——, discussion on hæmatosalpinx complicating myoma, Obst. 123

—— —— on melanotic tumour of vulva, Obst. 126

—— —— on œdematous fibroma of pelvis, Obst. 10

——, hæmatosalpinx with acute symptoms following retroversion of fibroid uterus, Obst. 171

——, ovarian dermoid retained two years in pelvis after obstructing labour, Obst. 35

Dore, S. E.—Discussion on case of scarlatiniform eruption following administration of quinine, Derm. 73

—— and Morris, Sir M.—Case of spreading telangiectases of the feet and legs, Derm. 89

Douche electrode for introducing current into rectum, Electr. 108

Dowsett, E. B.—Discussion on dental uses of paraform, Odont. 43

—— —— on difficult extraction, Odont. 139

—— —— on movements of mandible, Odont. 97

Drainage, satisfactory results in aiding suppression of malaria, Epid. 315, 316

——, surgical, in cure of pyo-pericardium (F. J. Steward and A. E. Garrod), Clin. 15

Dressing, paraform as, Odont. 41

Drew, Douglas.—Discussion on Wertheim's operation for carcinoma of cervix, Obst. 104

——, dislocation of the patella, ? congenital; operation, cure, Clin. 11

——, tuberculous synovitis of knee-joint in which arthrectomy was performed on two occasions, a movable joint resulting, Clin. 10

Drinkwater, on brachydactyly illustrating Mendelian heredity, quoted, Epid. 141

Dropsy of knee-joint, treatment by iodine and lithium ions, Electr. 81

Drugs, change of, importance of, in treatment of disease, Therap. 28

—— fail to cure rheumatism, Therap, 70, 75

Duckworth, Sir Dyce.—Discussion on case of angina pectoris with aortitis, Clin. 118

——, discussion on case of cerebellar tumour with proptosis, Clin. 128

—— —— on cases of gout showing destructive changes in bone, Clin. 230

—— —— on case of leucodermia with symptoms of Addison's disease, Clin. 212

—— —— on multiple rheumatic nodules in adult, Clin. 14

—— —— on multiple tumours of skin, Clin. 20

Dudgeon, L. S., results respecting bacterial invasion of peritoneum, alluded to, Path. 108

—— and Shattock, S. G.—Observations upon phagocytosis carried out by means of melanin, more especially with reference to the part taken by the leucocytes in infectious diseases, Med. 169-194

——, see also *Shattock, S. G.*, relationship between avian and human tuberculosis

Dukes, Clement, views on the "fourth disease," alluded to, Epid. 112, 124

Dumb-bell exercise, effect on blood-pressure, Therap. 36

Duncanson, J. Gray.—Discussion on arteriosclerosis, Therap. 38

——, discussion on development of hypnotism, Therap. 61

—— —— on nutmeg poisoning, Therap. 44

—— —— on rheumatism and chorea in childhood, Therap. 85

—— —— on therapeutics of indigestion, Therap. 20

—— —— on treatment of functional dyspepsia, Therap. 11

—— —— on tropical diseases and remedies, Therap. 28

Dunn, R. A., on Hertford epidemic, alluded to, Epid. 112

Dupuytren's contraction, treatment by electrolysis, Electr. 122

Dysentery, relation to ulcerative colitis, Med. 163-167

Dysmenorrhœa, membranous, treatment by ionic medication, Electr. 135

Dyspepsia, cooked vegetable and farinaceous food a cause of, Therap. 18

——, definition of, Therap. 3

—— (functional), causes, Therap. 4, 5

—— ——, classification of forms, Therap. 4

Dyspepsia (functional), complexity of disorders producing, Therap. 4
—— ——, due to organic disease, Therap. 9
—— ——, possibly followed by gastritis, Therap. 7
—— ——, treatment, discussion on, Therap. 3-12 (opened by Dr. Robert Hutchison, continued by Dr. Habershon, Dr. Herschell, Dr. H. C. Cameron, Dr. J. Gray Duncanson, Dr. Hutchison—reply)
—— (irritable), treatment, Therap. 15
—— —— —— by drugs, Therap. 17
—— —— ——, dietetic, Therap. 15
—— (nervous), treatment, Therap. 8
—— (somatic), nature of, Therap. 14
Dysphagia, reason for removal of goitre, Surg. 24
Dysphonia, reason for removal of goitre, Surg. 24
Dyspnœa in goitre, cause of, Surg. 26
—— reason for removal of goitre, Surg. 22
Dystrophia muscularis congenita, case of (R. Howard), Path. 157
—— —— —— —— association with congenital talipes equino-varus (R. Howard), Path. 157
—— —— —— —— microscopical examination of sections post mortem, Path. 162

Ear, discharges from, bacteria found in, Otol. 102-113, 119
—— ——, clinical pathology of (V. Wyatt Wingrave), Otol. 95-117
—— ——, collection of material, Otol. 97
—— ——, colour, Otol. 97
—— ——, composition, Otol. 99-113
—— —— ——, bacteria, Otol. 102
—— —— ——, cells, Otol. 99
—— —— ——, matrix, Otol. 113
—— ——, density, Otol. 96
—— ——, fœtor in, Otol. 96
—— ——, staining, Otol. 98
——, epithelioma of, two cases (H. Tod), Otol. 15
——, foreign body in (M. Yearsley), Otol. 6
——, lupus erythematosus affecting hands, ears and scalp (J. M. H. MacLeod), Derm. 88
——, tuberculosis of, Otol. 106, 119, 120
—— (middle), discharges, cytological examination (J. M. Darling), Otol. 163
—— ——, disease of, association with labyrinthitis, Otol. 53
—— ——, epithelioma of (traumatic ?) (W. Milligan), Otol. 91
—— ——, suppuration, acute, nature of aural discharge in, Otol. 114
—— —— ——, chronic, varieties of aural discharge in, Otol. 114, 115
—— —— ——, left temporo-sphenoidal abscess occurring in course of; patient exhibited after operation (A. Logan Turner), Otol. 137
—— —— ——, with phlebitis of lateral sinus and cerebellar abscess (P. McBride), Otol. 135
Eason, H. L.—Discussion on tumour of mediastinum, Clin. 95
East London Hospital for Children, statistics of complications of pneumonia at (1902-6) (A. M. Gossage), Med. 63, 66
Eccles, W. McAdam.—Discussion on complete gastrectomy, Surg. 92
Eclampsia of pregnancy, cerebral hæmorrhage as immediate cause of death in, Obst. 92-94
—— —— —— —— —— (into pons Varolii) as immediate cause of death in (N. C. Carver and J. S. Fairbairn), Obst. 79
——, lactic acid in urine and blood in, Path. 131
Ectropion of cervical mucosa in infant (R. Jamison), Obst. 323
Eczema, chronic, of both auricles and meatuses with unusual degree of hyperplasia (E. Furniss Potter), Otol. 132
Edelmann's whistle, use in hysterical nerve deafness, Otol. 128
Eden, T. W.—Case of primary bilateral papillary carcinoma of the ovary, Obst. 243; discussion (p. 244): Dr. Lockyer

Eden, T. W.—Chorionepithelioma of the uterus, with extensive vaginal and visceral metastases, Obst. 13; discussion (p. 17): Dr. H. R. Spencer, Mr. Malcolm

——, discussion on case of difficult labour, Obst. 149

—— —— on development of early human ovum, Obst. 277

—— —— on myomectomy during pregnancy, Obst. 137

—— —— on pregnancy in wall of tubal pus sac, Obst. 251

Edmunds, A.—Discussion on treatment of leukæmia, exophthalmic goitre and malignant disease by X-rays, Electr. 60

Edwards, F. **Swinford**.—Discussion on sciatica and its surgical treatment, Surg. 171.

—— —— on treatment of ulcerative proctitis by zinc cataphoresis, Surg. 185

Egg-laying, relation of blood-calcium metabolism to, Obst. 295, 296

—— —— —— ——, observations on hens respecting, Obst. 295, 296

Elastic pressure, continued (Aveling's method), in treatment of chronic inversion of uterus, Med. 283, 285, 288-290

Elbow-joint, flexion of, to acute angle, and slinging of arm to neck in treatment of fractured humerus, Surg. 222-225, 230

——, osteo-arthritis of, in case of old fracture of humerus (R. J. Godlee), Clin. 95

Electric baths in treatment of atonic conditions of digestive system, Electr. 110

—— current, circuit of, necessity of proportioning resistance of variable part to that of working part, Electr. 115

—— ——, see also *High frequency current*; *Interrupted currents*

—— machine, kind required in electrical treatment of atony of muscular coat, Electr. 105

—— machines for producing slow waves of current, Electr. 116

Electricity conduction in liquids, Electr. 67

—— —— in metals, Electr. 67

—— in treatment of atonic conditions of digestive system, Electr. 101

—— —— —— —— —— ——, special method for cases of true atony of muscular coat, Electr. 102-105

—— in treatment of chronic constipation, Electr. 101, 108, 109

——, sleep produced by, Electr. 11

——, static, in treatment of sciatica, Surg. 174, 175

——, value of, in treatment of dyspepsia, discussed, Therap. 7, 10

Electrodes, forms preferable in treatment of pelvic disease by ionic medication, Electr. 136, 137

——, number and method of application in treatment of atony of muscular coat, Electr. 104

Electrolysis in treatment of aneurysm, Electr. 124

—— —— —— of ankylosis, Electr. 122

—— —— —— of Dupuytren's contraction, Electr. 122

—— —— —— of sclerosis of spinal cord, Electr. 123

—— and excision, thyro-lingual fistula treated by (J. Dundas Grant), Laryng. 88

——, laws of, Electr. 68

——, principles of, Electr. 129, 131

Electrolytic method of measuring X-rays (H. Pirie), Electr. 92

Electro-therapeutics, applications by means of arm-baths, Electr. 115

——, future of (W. Deane Butcher), Electr. 1

——, interrupted currents in, value of, Electr. 114

——, progress in, Electr. 6

——, rhythmical currents in, Electr. 114, 115

——, see also under *Electricity*, *Electrolysis*; *Enemata Electrica*, *I*[illegible]

Elephantiasis græcorum, cases (H. Radcliffe Crocker and G. Pernet), Derm. 3

Elliot, A. M.—Discussion on acute ulcerative colitis, Med. 165

Elliotson, John, mesmeric experiments and researches of, Therap. 49

—— —— —— —— —— lead to his ruin, Therap. 50

Embolism, pulmonary, accompanied by convulsions, Med. 97

Emerson on classification of cases of leucocythæmia, quoted, Clin. 133
Emigration from Ireland, Epid. 10
Emotional temperament and nervous instability paramount factors in causation of chorea, Therap. 72
Empyema, chronic bilateral, of frontal, ethmoidal and sphenoidal sinuses, operative treatment, case showing result (H. Tilley), Clin. 196
—— —— of left frontal sinus; case shown two years after simplified Killian's operation (H. Tilley), Laryng. 35
—— complicating pneumonia, Med. 6, 33, 34, 38, 49
—— of mastoid (Bezold's) discharging into pharynx (W. S. Syme), Otol. 125
——, statistics of incidence after pneumonia at different ages, Med. 66
Encephalocele through posterior fontanelle (tumour of fœtal head) (H. S. Sington), Obst. 158
Endocarditis complicating pneumonia, Med. 9, 39
——, subcutaneous nodules in rheumatism associated with, Therap. 67
—— (malignant), recovery from, after injections of vaccine, Med. 15
—— (pneumococcal), complicating pneumonia; death from embolism of coronary arteries (case referred to in discussion), Clin. 118
—— (tuberculous), two cases of (W. O. Meek), Path. 116
—— —— —— —— causation of lesion proved to be due to tubercle bacillus in first, but not in second case, Path. 116, 122
Endothelial cells, origin of, Path. 169
Endothelioma of lower jaw, treatment by X-rays (W. Ironside Bruce), Electr. 59
—— involving lower part of larynx and pharynx (H. B. Robinson), Laryng. 68
——, removal from hard palate (D. McKenzie), Laryng. 62
Endotheliomata, reasons for enlargement of group, Path. 172
——, relations to other forms of new growth, Path. 167
——, variability of, Path. 172
Enema, daily use of, in treatment of chronic constipation, Med. 126
——, importance of use before treatment with purgatives, Med. 146
Enemata (electric) in treatment of colitis, Surg. 177
Energy, transformation of, by Röntgen ray tube, Electr. 93
England birthplace of hypnotism, Therap. 49
——, disappearance of malaria from, how accounted for, Therap. 24
Enteroptosis, cause of, Obst. 199
—— with chronic constipation, Med. 142
Entheliomata, Path. 168
Environment, differences of, producing differences amongst bacteria, Path. 72, 73
Epidemics, individual, analysis of, Epid. 37
——, see also *Influenza*, *Measles*, *Plague*, *Rubella*, *Small-pox*, *Typhoid Fever*, *Typhus Fever*
Epiglottis, lupus of, case (W. Jobson Horne), Laryng. 106
——, new instrument for passing suture through (C. Horsford), Laryng. 95
——, ulceration and destruction of (W. Jobson Horne), Laryng. 33
Epiglottis holders (Sir F. Semon), Laryng. 41
Epiphysis, separation of, causing coxa valga, Surg. 112
Epilepsy, association of migraine with, Med. 89
—— —— —— of Raynaud's disease with, Med. 87, 88
——, blood-pressure in, Med. 83
——, cardiac inhibition in, Med. 82
——, cerebral intravascular clotting as cause of, Med. 93
——, connection of infantile convulsions with, Med. 91
——, fits of, association of Raynaud's disease with, Med. 88
—— —— ——, correlation between circulatory changes in, and symptoms and recovery from attack, Med. 94
—— —— ——, physiological mechanism underlying recovery from, Med. 98
—— —— ——, symptoms, Med. 96-98

Epilepsy, instability of vasomotor system in, Med. 82
——, mental emotion as excitant of, Med. 92
——, pathology of (Alfred E. Russell), Med. 72
——, tremor of right arm associated with (F. E. Batten), Neur. 54
——, vagal and vaso-vagal attacks in, Med. 84
——, vasomotor spasm in brain as cause of, Med. 87
—— (idiopathic), evidence of cardiac arrest in, Med. 76
—— (minor), Med. 90
—— ——, association of bradycardia with, Med. 90
—— ——, syncopal attacks passing into, Med. 80
Epileptics, lactic acid in urine of, Path. 132
Epileptiform seizures of general paralysis, Med. 108
Epistaxis in childhood followed by bilateral telangiectases of trunk in later life (T. Colcott Fox), Derm. 66
—— in rheumatism, Therap. 67
—— (recurrent), accompanying multiple hereditary developmental angiomata (telangiectases) of skin and mucous membranes (F. Parkes Weber), Clin. 65
—— ——, dangerous attacks of, caused by localized pulsating growth of middle turbinate body (A. Tweedie), Laryng. 1
Epithelial cells in aural discharges, Otol. 99
Epithelioid cells, see *Mesothelial cells*
Epithelioma following Röntgen ray treatment of lupus, Electr. 121
—— of left auricle, with secondary involvement of auditory canal (H. Tod), Otol. 16
——, very extensive, of fauces (J. Dundas Grant), Laryng. 64
—— of middle ear (traumatic) (W. Milligan), Otol. 91
—— of tongue, treatment by X-rays (W. Ironside Bruce), Electr. 59
—— —— and fauces, case shown after operations (W. Chichele Nourse), Laryng. 56
—— of left tonsil, faucial pillar and uvula (J. Dundas Grant), Laryng. 29
—— (primary) of tympanic cavity, with secondary infection of skin over mastoid region (H. Tod), Otol. 15
—— of tympanum following chronic suppurative otitis media (A. L. Whitehead), Otol. 34
—— of left vocal cord (H. Tilley), Laryng. 60
Epitheliomatous degeneration of lupus scars after X-ray treatment, Electr. 87
Ergot, value of, in reducing gastric secretion, Therap. 9
Eristalis arbustorum and *dimidiatus*, larvæ of, cases reported of passage through intestine, Path. 151
—— *tenax*, larvæ of, passed by bowel; three specimens exhibited (S. G. Shattock), Path. 147
—— ——, presence in bowel probably due to ingestion of watercress containing ova, Path. 148, 149
Eruption (incomplete) of temporary molars (H. W. Trewby), Odont. 99
—— of extremities, case for diagnosis (G. W. Dawson), Derm. 25
—— (scarlatiniform, acute) following administration of small doses of quinine (J. Galloway and Dr. Cohen), Derm. 71
——, see also *Bromide* (eruption following administration of); *Ringed eruption*
Erythema, case (T. J. P. Hartigan), Derm. 14
—— (circinate) of two years duration in boy (H. G. Adamson), Derm. 10
—— elevatum diutinum, relation of granuloma annulare to, Derm. 149
—— induratum, see *Bazin's* disease
—— multiforme in rheumatism, Therap. 67
Erythematous eruption of unusual type (T. Colcott Fox), Derm. 176
—— syphilide (circinate), case of (A. Shillitoe), Derm. 21
Erythroblasts in aural discharges, Otol. 102
Erythromelalgia and splenic polycythæmia, Clin. 41, 42
Esch, his case of cerebral hæmorrhage in eclampsia of pregnancy described, Obst. 88
Esdaile, James, encouraged in his mesmeric work in India; discouraged on his return to Scotland, Therap. 51

Esdaile, James, painless operations under mesmerism by, Therap. 50, 51
Esox lucius, see *Pike*
Ether, anæsthesia by, in operation for goitre, Surg. 79
Ethmoidal cell (anterior), mucocele of left side of, opened from nose (D. McKenzie), Laryng. 101
—— ——, see also *Orbito-ethmoidal cells*
—— sinus; bilateral empyema of frontal, ethmoidal and sphenoidal sinuses: operative treatment, case showing result (H. Tilley), Clin. 196
Eudiometers, use of, in estimating reverse current passing through X-ray tube, Electr. 96, 97
Eustachian tube, injury to pharyngeal portion, from operative procedures (W. S. Syme), Otol. 126
Evans, Arthur.—Case of diffuse fibromata of the gums, with pathological report by Dr. J. Bernstein, Odont. 125, 127; discussion (p. 128): Mr. Baldwin, Dr. A. W. Baker, Mr. J. F. Colyer; Mr. Evans (reply)
——, specimen of leprosy of the larynx, Laryng. 16
Evans, Willmott.—Discussion on case of scarlatiniform eruption following administration of quinine, Derm. 71
Evolution in infectious diseases, Epid. 131
——, predominance of the prepotent a factor in, Odont. 120
—— —— —— —— ——, illustrated by breeding of pigeons, Odont. 120
Ewart, William.—Discussion on acute suffocative catarrh of Laennec, Med. 159
Ewing and Wolf, analyses of urine in pregnancy, Path. 134, 136, 142
Exercise cause of increase of temperature in pulmonary tuberculosis, Med. 249, 251
—— in treatment of choreic ataxy, Therap. 76
Exophthalmos in case of cerebellar tumour (J. P. Parkinson and J. S. Hosford), Clin. 124
Exostoses developing in connection with other bones after formation of diffuse cancellous osteoma of femur following fracture, Path. 83-94, 109
Expectoration, habit of, among men, factor in spread of pneumonia, Med. 49
Exposure, bodily, in causation of pneumonia, Med. 35, 41
Extension treatment of fracture of neck of femur, reasons for exercise of care in, Surg. 134
Extermination of species as a means of combating disease, Therap. 27
Extinction, "tendency" of certain diseases towards, Epid. 26
Extra-systoles following administration of digitalis, Med. 262
Extremities, eruption of; case for diagnosis (G. W. Dawson), Derm. 25
—— (upper and lower), post-mortem description in case of congenital defect of muscular system, Path. 157-160
Extremity (lower), arteritis obliterans of, with intermittent claudication ("angina cruris") (F. Parkes Weber), Clin. 44
—— (upper, right), arteritis obliterans of, associated with malformation of both clavicles (L. Guthrie), Neur. 101
—— —— ——, ischæmic disturbance of (F. Parkes Weber and E. Michels), Neur. 100
Eye-ball, muscles of, see *Muscles* (ocular)
Eye-blinking a habit tic, Neur. 41, 42
Eyes, colour of, illustrating Mendelism, Epid. 141
——, ulceration of, and phlyctenular conjunctivitis in association with well-marked rosacea (E. G. Graham Little), Derm. 181

Face, lupus erythematosus of, with condition of fingers simulating Raynaud's disease (J. H. Sequeira), Derm. 17
——, lymphangioma of (J. P. Roughton), Clin. 91
——, lymphangitis of, chronic, with disseminated lupus (A. Whitfield), Derm. 93
——, œdema of, chronic (W. S. Fox), Derm. 13
——, pigmentation of (G. Pernet), Derm. 17
——, post-mortem dissection of, in case of congenital defect of muscular system, Path. 159
——, vegetating granulomata on (T. Colcott Fox), Derm. 25
——, xanthoma of, multiple, of diabetic type, in infant (F. Parkes Weber), Derm. 92
——, see also *Dento-facial* irregularities

Fagge, C. H.—Aneurysm of the common femoral artery; excision of aneurysm and common femoral vein, Clin. 232

——, branchial sinus leading into external auditory meatus; sinus excised, Otol. 90; discussion (p. 91): Mr. Yearsley

——, case in which, for recurrent sarcoma, parts of the femoral artery and vein were excised, Clin. 231

——, discussion on case for diagnosis, Otol. 27

—— —— —— of Menière's disease, Otol. 26

—— —— —— of rupture of brachial plexus at birth, Clin. 219

—— —— on keloid following mastoid operation, Otol. 21

—— —— on operative surgery of labyrinthitis, Otol. 79

—— —— on pulsating growth in left external auditory meatus, Otol. 33

Fagge, C. Hilton, on epileptic aura, quoted, Med. 95

Fairbairn, J. S.—Rupture of an early tubal gestation causing death within a few hours, Obst. 37; discussion (p. 40): Dr. A. Giles

—— and Carver, Norman C.—Hæmorrhage into the pons Varolii as the immediate cause of death in eclampsia, with illustrative cases, Obst. 79; discussion (p. 92): Dr. Herbert Spencer, Dr. Macnaughton Jones, Mr. A. Lionel Smith, Dr. Amand Routh, Mr. D. C. Rayner, Dr. R. H. Hodgson, Dr. Fairbairn (reply)

Fairbank, H. A. T.—Sarcoma of thigh, Clin. 109

Fallopian tube, encapsulation of sterile fluid in connection with (J. Bland-Sutton), Obst. 229

—— ——, tuberculosis of, primary unilateral (H. Macnaughton Jones), Obst. 177

—— ——, twisted inflamed specimen exhibited (J. D. Malcolm), Obst. 99

—— ——, see also *Pregnancy* (tubal)

Farabee on brachydactyly illustrating Mendelian heredity, quoted, Epid. 141

Farinaceous food, cooking of, untoward effects on jaws, teeth, and digestion, Therap. 48

Fascia, pelvic, anatomy of, Obst. 52

Fasciæ, nature of, Obst. 52

Fat, diet rich in, valuable in treating hyperchlorhydria, Therap. 11

Fatty degeneration in arteriosclerosis, Therap. 33

Fauces, epithelioma of, very extensive (J. Dundas Grant), Laryng. 64

—— —— of tongue and fauces, case shown after operations (W. Chichele Nourse), Laryng. 56

—— (pillar of): epithelioma of left tonsil, faucial pillar and uvula (J. Dundas Grant), Laryng. 29

Favus in mouse (T. P. Beddoes for P. S. Abraham), Derm. 64

—— of scalp, two cases (E. G. Graham Little), Derm. 26

——, treatment by Röntgen rays contraindicated in children, Electr. 119-120

Fawcett, Professor Edward, on development of maxilla and mandible, quoted, Odont. 17, 19, 20

Fawcett, John.—Discussion on pneumonia and its complications, Med. 44

——, pneumothorax treated by aspiration under the X-rays, Clin. 38; discussion (p. 39): Dr. Parkes Weber, Dr. G. A. Sutherland, Dr. Herringham, Dr. Fawcett (reply)

——, Mann, H. C. C., and Cameron, H. C.—Statistics of complications of pneumonia from Guy's Hospital (1902-6), Med. 69

Femoral ring, cure of ascites by permanent drainage through (W. E. Wynter), Clin. 49

Femur, fracture and malunion of, followed by right coxa valga, Surg. 129

—— —— of neck of, treatment by extension, reasons for exercise of care in, Surg. 134

——, head of, perforation of acetabulum by: skiagram showing (H. Lewis Jones), Electr. 34

——, osteoma of, diffuse cancellous, following fracture (W. H. Battle and S. G. Shattock), Path. 83-94

—— —— —— ——, histology, Path. 96

—— —— —— ——, pathological account and remarks (S. G. Shattock), Path. 94-114

—— —— —— ——, spontaneous disappearance, Path. 109

——, region of, skiagram of peculiar injury in, with perforation of acetabulum by head of femur (H. Lewis Jones), Electr. 34

——, sarcoma of (H. A. T. Fairbank), Clin. 109

Femur, sarcoma of, recurrent; excision of parts of femora artery and vein (C. H. Fagge), Clin. 231
——, tumour in (C. W. Rowntree), Clin. 20
Fenestra ovalis, Otol. 45
Ferrand and Lemaine, on the tuberculin cutaneous and ophthalmic reactions, quoted, Path. 76
Ferrier, David, F.R.S.—Discussion on case of dog suffering from ataxia, Neur. 26
——. discussion on mechanism of nystagmus, Neur. 79
—— —— on pathology of epilepsy, Med. 112
—— —— on tumours of fourth ventricle, Neur. 36
Fertility, menstruation not a causal factor of, Obst. 294
Fever, "eruptive," small-pox mistaken for, in Trinidad epidemic, Epid. 232
Fibroid tumour (cervical) complicating pregnancy; subtotal hysterectomy at fourth month (J. Bland-Sutton), Obst. 17
—— —— in state of "red degeneration" in pregnant uterus (C. E. Purslow), Obst. 42
—— —— of uterus, with pyosalpinx due to traumatism, Obst. 123
—— tumours complicating pregnancy, case showing "red degeneration" (F. J. McCann), Obst. 315
—— —— —— ——, treatment, methods of, Obst. 316-319
—— ——, rupture of uterus in presence of, Obst. 131, 137, 138
—— —— of uterus showing sarcomatous degeneration (W. C. Swayne), Obst. 151
—— —— ——, report of Pathology Committee on, Obst. 151
Fibroma of naso-pharynx simulated by large post-nasal polypus growing from antrum of Highmore (J. Dundas Grant), Laryng. 102
—— ——, pedunculated, large (fibromyxoma), removed from vault of naso-pharynx of boy of 9 (H. J. Davis), Laryng. 138
—— (œdematous) of pelvis (W. Tate), Obst. 7
—— —— of left vocal cord; removal; patient shown (C. Horsford), Laryng. 24
Fibromata of gums, diffuse (A. Evans), Odont. 125
—— of skin with developing neuro-fibroma (D. Walsh), Derm. 189
Fibromyoma of uterus, enucleation during seventh lunar month of pregnancy (H. Williamson), Obst. 73
—— (sarcoma) of uterus (J. Bland-Sutton), Obst. 233
—— (paravaginal), removal and exhibition of specimen (F. J. McCann), Obst. 321
—— (retroperitoneal), subligamentous, large, complicating pregnancy (J. H. Dauber), Obst. 132
Fibro-psammoma of cerebellum with proptosis, Clin. 126
Fibrous constriction of trachea (H. Tilley), Laryng. 35
Fibula, congenital absence of, and outer half of foot (T. H. Openshaw), Clin. 9
—— and hyoid bone affected by leontiasis ossea, Path. 93
—— and tibia, hypertrophied callus of, Path. 101
Filaria, curative drug treatment of, undiscovered, Therap. 26
——, extra-corporeal life in Culex mosquito, Therap. 24, 25
—— —— —— —— —— —— basis of mosquito-malaria theory, Therap. 24, 25
Filatow on "fourth disease," quoted, Epid. 112, 116
Fingers, clubbing of fingers and toes with caries of spine (P. M. Heath), Clin. 234
——, condition of, simulating Raynaud's disease in case of lupus erythematosus of face (J. H. Sequeira), Derm. 17
Finny, on epileptic attacks associated with failure of pulse, quoted, Med. 75, 76
—— on petit mal and bradycardia, quoted, Med. 90
Finsen light treatment of lupus vulgaris, Electr. 85
Finzi, N. S.—Discussion on electrolytic method of measuring X-rays, Electr. 99
Firth, Col.—Discussion on etiology and epidemiology of typhoid fever, Epid. 221
Fishes, see *Fossil* fishes
Fistula (median cervical); case shown after operation (J. Dundas Grant and D. McKenzie), Laryng. 63

Fistula (thyro-lingual) treated by electrolysis and excision (J. Dundas Grant), Laryng. 88
Fistulous opening in gingivo-labial recess in case of suppurating hæmatoma of septum nasi (D. McKenzie), Laryng. 92
—— —— in middle line of neck just above hyoid bone (D. McKenzie), Laryng. 3
Flatulence remedied by carminatived asafœtida, Therap. 19
Fleas, agency of, in spreading plague, Epid. 74, 75
——, destruction of, as means of plague prevention, Epid. 74, 83, 89, 94
Fleetwood, cause of outbreak of typhoid in Militia battalion in camp at, Epid. 222
FLETCHER, H. MORLEY.—Discussion on amyotonia congenita, Clin. 49
——, discussion on pathology and treatment of constipation, Med. 148
Flour (white, roller-milled) cause of dental decay, Odont. 71, 72
Fluid (sterile), encapsulation of, in connection with Fallopian tube and ovary (J. Bland-Sutton), Obst. 229
Fluids, ingestion of, prejudicial effect in gastric motor insufficiency, Therap. 7
Fœtal head, tumour of (encephalocele through posterior fontanelle) (H. S. Sington), Obst. 104, 158
Fœtor in aural discharges, Otol. 96
Fœtus, death of, *in utero*, Obst. 147, 148
—— —— ——, occurring in six consecutive pregnancies (H. Williamson and E. L. Holland), Obst. 161
—— —— ——, syphilis cause of, Obst. 161
—— —— —— —— —— proved by demonstration of presence of *Spirochæta pallida* in tissues, Obst. 162
——, saving life of, in cases of pregnancy complicated by fibroid tumour, Obst. 320
——, post-maturity of, with excessive development, Obst. 146
——, tissues of, importance of examination for presence of *Spirochæta pallida* in (H. Williamson and E. L. Holland), Obst. 161-163
—— (congenital syphilitic), constant presence of *Spirochæta pallida* in organs of, evidence of its being specific organism of syphilis, Obst. 167
Folin, analyses of excretion under normal diet quoted, Path. 138
Folliculitis (conglomerate), see *Horse ringworm*, pustular
—— decalvans (pseudo-pelade of Brocq) (E. G. Graham Little), Derm. 86, 165
Fontanelle, posterior, encephalocele through (tumour of fœtal head) (H. S. Sington), Obst. 158
Food, influence of, on production of dental caries, Odont. 73, 114, 123
——, obtaining, by artificial means first great stage in evolution of human diet, Therap. 45
——, pollution of, cause of spread of typhoid fever, Epid. 171, 172, 187, 188
——, preparation by artificial means (pounding and cooking) second great stage in evolution of human diet, Therap. 45, 46
——, production of, by artificial means third great stage in evolution of human diet, Therap. 46; see also *Cibiculture*
Foodstuffs requiring efficient mastication, importance of, in early years in prevention of dental caries, Odont. 37
Foot [or feet], congenital absence of fibula and outer half of foot (T. H. Openshaw), Clin. 9
—— ——, feet and hands only affected in case of lupus erythematosus (H. G. Adamson), Derm. 2
—— ——, œdema of hands and feet with mediastinal affection (F. Parkes Weber), Clin. 192
—— ——, telangiectases (spreading) of feet and legs (Sir M. Morris and S. E. Dore), Derm. 89
——, hæmangiectatic hypertrophy of (F. Parkes Weber), Derm. 59
—— —— —— —— possibly of spinal origin (F. Parkes Weber), Clin. 193; Neur. 19
FORBES, J. GRAHAM.—Case showing unusual situation of gouty deposit in the olecranon bursa, Clin. 53; discussion (p. 54). Prof. Osler, Dr. Poynton, Dr. Parkes Weber, Dr. A. E. Garrod
——, congenital absence of left pectoral muscles, Clin. 71; discussion (p. 74): Mr. Raymond Johnson, Dr. G. A. Sutherland; Dr. Forbes (reply)
——, discussion on case of syphilitic pachydermia, Laryng. 48
——, report on case previously shown of unusual gouty deposit of olecranon bursa, Clin. 80

FORBES, J. GRAHAM, and BATTEN, F. E.—Note on two cases of Gaertner infection (*Bacillus enteritidis*) in infants, Clin. 81
—— and LANGMEAD, FREDERICK S.—Fatal lymphocythæmia in early life, Clin. 129-177; discussion (p. 167): Sir T. Barlow, Dr. Parkes Weber, Dr. Poynton, Dr. Forbes (reply), Dr. Langmead (reply)
FORBES-ROSS, F. W.—Discussion on arterio-venous anastomosis, Clin. 208
—— —— on vaccine treatment by the mouth, Med. 239
Forceps delivery followed by coxa valga, Surg. 112
—— (laryngeal), universal, for use by direct and indirect methods (P. Watson Williams), Laryng. 53
Ford, experiments respecting transport of bacteria to viscera, quoted, Path. 108
Forearm (right), annular lichen planus affecting penis and (J. M. H. MacLeod), Derm. 34
Forehead (high congenital), spurious acromegaly in patient suffering from exophthalmic goitre associated with (D. Walsh), Clin. 195
Foreign body in ear (M. Yearsley), Otol. 6
Fossil fishes, teeth of (J. Humphreys), Odont. 7
Fossula rotunda, Otol. 44
FOTHERGILL, W. E.—The supports of the pelvic viscera: a review of some recent contributions to pelvic anatomy, with a clinical introduction, Obst. 43; discussion (p. 58): Dr. Amand Routh, Dr. R. H. Paramore, Dr. Briggs, Sir Arthur Macan, Dr. H. Spencer
——, discussion on the supports of the pelvic viscera, Obst. 218
FOULERTON, A. G. R.—Discussion on complications of pneumonia, Med. 15
"Fourth disease," Epid. 112, 113-117, 120
— ——, supposed distinctions from scarlet fever and rubella, Epid. 114, 115
Fowl (comb-bearing), crossing of Breda fowl with, illustration of nature of alternative pairs of characters, Epid. 148
—— (domestic), incomplete glandular hermaphroditism in (S. G. Shattock and C. G. Seligmann), Path. 3
Fowls, secondary sexual characters of, influence of testis upon (C. E. Walker), Path. 153
FOX, H. CLAYTON.—Anosmia, dryness and crustings of the nose in a man aged 30, Laryng. 13
——, case of laryngeal paralysis, Laryng. 107; discussion (p. 107): Dr. Vinrace, Mr. Barwell, Dr. FitzGerald Powell, Dr. Grant, Mr. Clayton Fox (reply)
——, discussion on bleeding polypus of septum, Laryng. 29
—— —— —— on case after operation for angina Ludovici, Laryng. 130
—— —— —— of infiltration of vocal cord, Laryng. 84
—— —— —— of necrosis of the arytænoid cartilage, Laryng. 65
—— —— —— of symmetrical nodules on cords, Laryng. 19
—— —— —— of syphilitic pachydermia, Laryng. 49
—— —— —— of tumour of vocal cord, Laryng. 9
—— —— —— of unilateral paralysis of tongue, Laryng. 54
—— —— on cases after the radical maxillary antrum operation, exemplifying simplification of the after-treatment, Laryng. 59
—— —— on cyst on floor of nose, Laryng. 97
—— —— on extreme form of septal deflection, Laryng. 6
—— —— on fistulous opening in middle line of neck, Laryng. 4
—— —— on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 104
—— —— on lipoma or cystoma of neck, Laryng. 39
—— —— on paresis of right vocal cord, Laryng. 2
—— —— on subcutaneous induration of the neck of uncertain origin, Laryng. 112
—— —— on subglottic laryngeal tumour, Laryng. 91
—— —— on value of X-rays in sounding and washing out frontal sinus, Laryng. 87
FOX, T. COLCOTT.—Case of bilateral telangiectases of the trunk, with a history of marked epistaxis in childhood and recent rectal hæmorrhage, Derm. 66; discussion (p. 69): Dr. Radcliffe Crocker, Dr. Colcott Fox (reply)

Fox, T. Colcott.—Case of coccogenic sycosis, Derm. 11
——, discussion on cases of elephantiasis graecorum, Derm. 55
—— —— on case of erythema induratum (Bazin) in which a positive ophthalmo-tuberculin reaction had been obtained, Derm. 31
—— —— —— of molluscum contagiosum, Derm. 182
—— —— —— of multiple telangiectases, Clin. 65
—— —— —— of nodular ringed eruption, Derm. 82
—— —— —— of tuberculides in girl aged 11 years, Derm. 175
—— —— —— of varus nodulosus of Brooke, Derm. 86
—— —— on dermatitis herpetiformis of unusual type, Derm. 180
—— —— on granuloma annulare, Derm. 162
—— —— on parakeratosis variegata, Derm. 176
—— —— on prurigo of Hebra, Derm. 182
——, erythematous eruption of unusual type, Derm. 176
——, vegetating granulomata on the face, Derm. 25
Fox, Wilfrid S.—Case for diagnosis, Derm. 70; discussion (p. 70): Dr. Radcliffe Crocker, Dr. Pringle, Dr. Whitfield
—— case of chronic œdema of the face, Derm. 13
—— —— of lichen plano-pilaris, Derm. 45
—— —— of verrucose nævus, Derm. 12
—— —— of X-ray dermatitis, Derm. 177; discussion (p. 177): Dr. Walsh
—— and Morris, Sir M.—Case of von Recklinghausen's disease, Derm. 16
Fracture in carpus, diagnosis by X-rays (A. D. Reid), Electr. 62
—— of femur followed by diffuse cancellous osteoma, similar growths afterwards developing in connection with other bones (W. H. Battle and S. G. Shattock), Path. 83-114
—— of humerus, sarcoma arising at site of, Path. 100
—— ——, severe compound (Rushton Parker), Surg. 221
—— ——, ununited, cases of (Rushton Parker), Surg. 232
—— through temporal bone, specimen illustrating (A. Cheatle), Otol. 10
Fractures, swelling of callus formed after, Path. 109
—— of scaphoid bone, diagnosis by X-rays (G. Harrison Orton), Electr. 62; (W. Y. Somerville) Electr. 63
—— (ununited), diagnosis of union by radiography, Surg. 222, 228, 235, 237
—— ——, treatment, Surg. 232-234, 236
—— —— ——, palliative, Surg. 236
Frambœsia, senile warts developing into fungating growths (frambœsiform) (H. Radcliffe Crocker and G. Pernet), Derm. 56
France, study of hypnotism in, commenced later than in England. Therap. 49, 53
Fraser, J. S.—Patient after vestibulotomy (right ear), Otol. 149. [For discussion see *Gibson*, G. and Lake, R.—notes of a case of ablation of both vestibules, &c.]
Freeman W. S.—Keratosis; case for diagnosis, Derm. 14
Fremantle, F. E.—Discussion on Mendelism in relation to disease, Epid. 166
——, discussion on plague, Epid. 95
French, Herbert S.—Case of multiple subcutaneous rheumatic nodules, Clin. 75
——, discussion on the therapeutics of indigestion, Therap. 21
——, tumour of mediastinum (? hydatid cyst), Clin. 93; discussion (p. 95): Dr. Eason, Dr. Theodore Williams, Dr. Cyril Ogle
French observers' work in study of tics, Neur. 44, 45
Friedreich's disease (G. Ogilvie), Neur. 92
Fright as an agent in causation of chorea compared with traumatism or chill, Therap. 72
——, effects on children, Med. 92
Frontal lobe (left cerebral), abscess in, originating from suppuration in left frontal sinus (L. V. Cargill, W. Turner and StClair Thomson), Laryng. 125
—— sinus, bilateral empyema of frontal, ethmoidal and sphenoidal sinuses, operative treatment; case showing result (H. Tilley), Clin. 196

Frontal sinus, disease of, treatment by Killian's complete and incomplete operation and Ogston-Luc operation ; stereoscopic photographs of patients (W. S. Syme), Laryng. 99
—— ——, distension (mucocele) of (F. J. Steward), Laryng. 71
—— —— ——, causing proptosis (StClair Thomson), Laryng. 49
—— —— —— —— ——, question of operation, Laryng. 50
—— ——, results of double operation on (StClair Thomson), Laryng. 13
—— ——, suppuration in, chronic ; treatment by Killian's operation (J. Dundas Grant), Laryng. 113
—— ——, value of X-rays in sounding and washing out (StClair Thomson), Laryng. 86
—— —— (left), empyema, chronic ; case shown two years after simplified Killian's operation (H. Tilley), Laryng. 35
—— —— ——, suppuration in, origin of abscess in left cerebral ontal lobe (L. V. Cargill, W. Turner and StClair Thomson), Laryng. 125
—— —— sinusitis (bilateral), case after operation (W. Chichele Nourse), Laryng. 25
Froriep, on etiology of congenital absence of pectoral muscles, quoted, Clin. 73

Gabell, D. P.—Discussion on classification of dento-facial irregularities, Odont. 144
——, discussion on dental uses of paraform, Odont. 43
—— —— on movements of mandible, Odont. 97
—— —— on teeth of fossil fishes, Odont. 15
Gaertner infection (*Bacillus enteritidis*) in infants (F. E. Batten and J. G. Forbes), Clin. 81
Gait as test for presence of labyrinthitis, Otol. 70
—— in coxa valga, Surg. 135
Galabin, A. L.—Discussion on ovarian dermoid with secondary cysts connected with omentum, Obst. 117
Galeazzi, description of cases of coxa valga quoted, Surg. 116
——, treatment of coxa valga by linear osteotomy ; method and results quoted, Surg. 138
Gall-bladder, lesions of, rare during attack of typhoid fever, Epid. 219
——, excision of, see *Cholecystectomy*
Galloway, James.—Case of extensive psoriasis with suppurating lesions, Derm. 41
——, dermatitis herpetiformis of unusual type, Derm. 177 ; discussion (p. 180) : Dr. C. Fox, Sir M. Morris, Dr. Whitfield, Dr. Galloway (reply)
——, discussion on case for diagnosis (granuloma of mouth with penile ulcer), Derm. 58
—— —— —— for diagnosis (? leprosy), Derm. 53
—— —— —— of (?) dermatitis artefacta, Derm. 85
—— —— —— of lupus with unusual features, Derm. 78
—— —— —— of ringed eruption, Derm. 48
—— —— —— —— on hand, Derm. 65
—— —— on granuloma annulare, Derm. 161
—— and Cohen, Dr.—Case of acute scarlatiniform eruption following the administration of small doses of quinine, Derm. 71 ; discussion (p. 73) : Dr. Dore, Mr. Willmott Evans, Dr. Radcliffe Crocker
Gall-stones, recurrence rare after cholecystectomy, Surg. 97, 98, 106
Galton's whistle, use in hysterical nerve deafness, Otol. 128, 129
Galvanism in treatment of atony of digestive system, Electr. 106, 107, 108
Galvano-cautery in treatment of lupus vulgaris, Electr. 84
—— and tracheotomy in treatment of extensive tuberculosis of larynx ; complete cicatrization (StClair Thomson), Laryng. 9
Gamete, definition and nature of, Epid. 136-138
Gametic coupling of hereditary characters, Epid. 151
Gangrene (senile), treatment of case by arteriovenous anastomosis (C. A. Ballance), Clin. 199
——, see also *Lung*, gangrene of
Garbage, removal of, results in disappearance of rats, Epid. 87, 88
Gardner, H. Bellamy.—Discussion on mechanism of nystagmus, Neur. 81

GARROD, A. E.—Case of multiple rheumatic nodules in an adult, Clin. 13; discussion (p. 14): Dr. S. West, Sir Dyce Duckworth, Dr. B. Abrahams, Dr. F. J. Poynton, Dr. Garrod (reply), Sir T. Barlow
——, discussion on case of angina pectoris with aortitis, Clin. 118
—— —— —— of myxœdema with unusual features, Clin. 29
—— —— —— of unusual situation of gouty deposit, Clin. 55
—— —— on congenital heart disease in adults, Clin. 37
—— —— on dislocation of patella with osteo-arthritis, Clin. 97
—— —— on Mendelism in relation to disease, Epid. 162
—— —— on ochronosis, Clin. 58
—— and STEWARD, F. J.—Case of pyo-pericardium cured by drainage, Clin. 15; discussion (p. 16): Dr. S. West, Mr. Godlee
Gasserian ganglion, excision of, for trigeminal neuralgia after unsuccessful intracranial neurotomy (J. Hutchinson, jun.), Clin. 21
Gastrectomy, complete, case of (B. G. A. Moynihan), Surg. 81
—— —— ——, pathological report (F. C. Moore), Surg. 88
Gastric secretion, hyperacidity in, diminution of, Therap. 5
—— —— —— ——, dietetic neutralization of, Therap. 5
—— —— reduced by ergot, Therap. 9
Gastritis, possible sequela of functional dyspepsia, Therap. 7
Gastro-intestinal symptoms in cases of lymphocythæmia, Clin. 160
Gastroschisis (K. Das), Obst. 323
——, description of specimen, Obst. 325
——, nomenclature and classification, Obst. 323
GEE, SAMUEL.—Discussion on acute suffocative catarrh of Laennec, Med. 158
——, discussion on acute ulcerative colitis, Med. 166
——, presidential address before Medical Section, Med. 1
Gelatine, value of, in treatment of hæmorrhage, Med. 247
Germ cells, variations in, Odont. 110
—— centres, Obst. 103
German measles, see *Rubella*
Gestation sac, complete, delivery of, at end of twenty-eighth week in case of placenta prævia, Obst. 314
Giant-cells in aural discharges, Otol. 102
Gibbings, A. T., on association of epileptic seizures with bradycardia, quoted, Med. 73
GIBSON, GEORGE, and LAKE, RICHARD.—Notes of a case of ablation of both vestibules for the relief of vertigo, Otol. 150; discussion (p. 154): Dr. W. Milligan, Dr. Albert Gray, Mr. Sydney Scott, Mr. Hunter Tod, Dr. Syme, Mr. A. L. Whitehead, Dr. Kerr Love, Dr. McBride, Dr. Fraser (reply)
Gibson, M. J., and Tweedy, E. Hastings, cases of pubiotomy alluded to, Obst. 22
Giemsa's stain for staining *Spirochæta pallida* in films, Obst. 164, 165, 167, 168
GILES, ARTHUR E.—Discussion on case of pregnancy complicated by large cervical fibroid, Obst. 18
—— discussion on myomectomy during pregnancy, Obst. 137
—— —— on rupture of early tubal gestation, Obst. 40
Gingivo-labial recess, fistulous opening in, in case of suppurating hæmatoma of septum nasi (D. McKenzie), Laryng. 92
Gland epithelium in aural discharges, Otol. 99
Glands, enlargement of, in cases of lymphocythæmia, Clin. 159
Glandular hermaphroditism, incomplete, in domestic fowl (S. G. Shattock and C. G. Seligmann), Path. 3
Glenoid cavity, excavation of, through posterior inter-muscular incision in reduction of old subcoracoid dislocation of humerus (W. G. Spencer), Surg. 187
—— ——, exposure at operation, Surg. 190, 193
Glossitis, case of (A. N. Leathem and J. M. H. MacLeod), Derm. 51

Gloucestershire, scheme of medical inspection in schools in use in (M. Coplans), Epid. 45

Gluck, remarks on performance of arterio-venous anastomosis, quoted, Clin. 204

Goadby, Kenneth W.—Discussion on case of extraction of upper wisdom tooth, Odont. 103

—— discussion on clinical notes on pyorrhœa alveolaris, Odont. 106

—— —— on heredity and dental disease, Odont. 122, 124

Goat, bones of, disease in, Path. 104

Godlee, R. J.—Discussion on lymphangioma of face, Clin. 92

—— discussion on multiple tumours of skin, Clin. 20

—— —— on pyo-pericardium cured by drainage, Clin. 16

——, old fracture of humerus with osteo-arthritis of elbow, Clin. 95

——, old-standing dislocation of patella, with osteo-arthritis of knee, Clin. 96; discussion (p. 96): Mr. Openshaw, Dr. Seymour Taylor, Dr. Poynton, Dr. Garrod, Mr. Godlee (reply)

Goitre complicated by hypertrophied thymus, Surg. 78

——, removal by operation, anæsthetic employed, Surg. 31

—— —— ——, analysis of 274 additional cases (J. Berry), Surg. 21-75

—— —— ——, complications after or during, Surg. 34

—— —— ——, drainage, Surg. 33

—— —— ——, position of patient, Surg. 32

—— —— ——, nature of cases, encapsuled, Surg. 25, 26, 27

—— —— —— —— ——, non-encapsuled, Surg. 25

—— —— —— —— of operation, enucleation, Surg. 29-31; extirpation, Surg. 29-31

—— —— ——, post-operative venous hæmorrhage dangerous, Surg. 32

—— —— ——, prevention of wound infection, Surg. 32

—— —— ——, reasons for, Surg. 22

—— —— ——, skin incisions, Surg. 32

——, situation of, Surg. 27

—— (adeno-parenchymatous), as represented in operated cases, Surg. 27

—— (exophthalmic), operation for, results, Surg. 79

—— —— ——, risks serious, Surg. 37

—— —— —— incomplete operation, results satisfactory, Surg. 37

—— ——, treatment by X-rays (W. Ironside Bruce), Electr. 57

—— ——, with spurious acromegaly in patient with congenitally high forehead (D. Walsh), Clin. 195

—— (intra-thoracic), non-malignant, as represented in operated cases, Surg. 28

—— (parenchymatous), as represented in operated cases, Surg. 26

—— ——, question of operation for, Surg. 76

Goldmann, Professor E.—The growth of malignant disease in man and the lower animals, with special reference to the vascular system, Surg. 1; discussion (p. 18): Sir William Church, Mr. Henry Morris, Dr. F. J. Smith, Prof. Goldmann (reply)

——, the value of X-rays in the diagnosis of obscure abdominal cases, Surg. 14

Golla, F. L.—Statistics of complications of pneumonia from St. George's Hospital (1897-1906), Med. 67

Gonococcus in aural discharges, Otol. 108

——, infection by, improvement under antistreptocococcic serum treatment, Med. 197

Gonorrhœal warts on penis, treatment by X-rays (A. D. Reid), Electr. 63

Goodall, E. W.—Discussion on etiology and epidemiology of typhoid fever, Epid. 218

——, rubella, Epid. 101; discussion (p. 121): Sir S. Murphy, Dr. Ransome, Dr. Caiger, Dr. M. Richards, Dr. F. N. Hume, Dr. Butler, Dr. Claude B. Ker, Dr. J. T. C. Nash, Dr. Goodall (reply)

Gorgas, Col. W. C., sanitary undertakings at Isthmus of Panama, Epid. 303, 306, 309, 310

Gossage, A. M.—Discussion on complications of pneumonia, Med. 32

——, discussion on pathology of epilepsy, Med. 114

——, statistics of complications of pneumonia from East London Hospital for Children (1902-6), Med. 63, 66

Gossage, A. M.—Statistics of complications of pneumonia from Westminster Hospital (1897-1906), Med. 61, 66

——, the tone of cardiac muscle (with references), Med. 253-270; discussion (p. 270): Dr. Lees, Dr. James Mackenzie, Professor Cushny, Dr. Alexander Morison, Dr. H. A. Caley, Dr. Gossage (reply)

Gould, A. Pearce. Discussion on case of hermaphroditism, Clin. 113

Gourmets, "over-educated" stomach of, Therap. 14

Gout, connection with pyorrhœa alveolaris, Odont. 104, 105

——, destructive changes in bone occurring in, cases showing (J. B. Burt), Clin. 223

Gouty deposit, unusual situation in olecranon bursa (J. G. Forbes), Clin. 53, 80

Gowers, Sir William R., F.R.S.—Discussion on intermittent claudication in lower extremity (angina pedis or angina cruris) from chronic arteritis obliterans, Neur. 51

——, the mechanism of nystagmus, Neur. 71; discussion (pp. 79-83): Dr. Ferrier, Dr. F. E. Batten, Mr. Ernest West, Dr. Charles Mercier, Mr. Bellamy Gardner, Mr. Sydney Scott, Dr. Henry Head, Sir W. R. Gowers (reply)

—— on association of migraine with epilepsy, quoted, Med. 89

—— on mental emotion as excitant of epilepsy, quoted, Med. 92

—— on symptoms of cerebral gummata, alluded to, Neur. 16, 18

—— on syncopal attacks passing into minor epilepsy, quoted, Med. 80

—— on vagal and vaso-vagal attacks, quoted, Med. 84

Grafting of nerve-root (P. Sargent), Neur. 8

—— (partial) with aid of local anæsthesia in after-treatment of radical mastoid operation (J. Stoddart Barr), Otol. 145, 148

Gram's stain, use in aural discharges, Otol. 98, 103, 104, 105, 106

Grant, J. Dundas.—Case of chronic suppuration of the maxillary antrum treated by operation through the inferior meatus, Laryng. 30; discussion (p. 30): Mr. Herbert Tilley, Dr. Watson Williams, Mr. Parker, Dr. Westmacott, Dr. StClair Thomson, Dr. J. B. Ball

——, case of congenital laryngeal stridor, Laryng. 109; discussion (p. 110): Dr. J. B. Ball, Dr. Grant (reply)

—— —— of epithelioma of the left tonsil, faucial pillar and uvula, Laryng. 29; discussion (p. 30): Mr. Nourse, Mr. Barwell

—— —— of extensive epithelioma of the epiglottis, Laryng. 80; discussion (p. 81): Mr. De Santi

—— —— of Killian's operation for chronic suppuration in the frontal sinus, Laryng. 113

—— —— of large post-nasal polypus simulating a naso-pharyngeal fibroma growing from the antrum of Highmore, Laryng. 102; discussion (pp. 103-106): Dr. J. B. Ball, Dr. P. McBride, Dr. FitzGerald Powell, Dr. StClair Thomson, Mr. Stuart-Low, Mr. Clayton Fox, Dr. Dan McKenzie, Mr. Kelson, Dr. Jobson Horne, Dr. Syme, Dr. Pegler, Dr. Grant (reply)

—— —— of lupoid ulcer of the floor of the vestibule of the nose, Laryng. 110; discussion (p. 110): Dr. J. B. Ball, Mr. Barwell, Dr. Grant (reply)

—— —— of lupus of the larynx and tonsil, Laryng. 78; discussion (p. 78): Mr. Barwell, Dr. Grant (reply)

—— —— of thyro-lingual fistula treated by electrolysis and excision, Laryng. 88

—— —— of unilateral nerve deafness in an elderly man, Otol. 130; discussion (p. 130): Dr. Purves Stewart, Mr. Yearsley, Dr. Milligan

—— —— of unilateral hysterical nerve deafness of sudden onset with hemi-anæsthesia and other allied stigmata, Otol. 127; discussion (p. 128): Dr. Purves Stewart, Dr. Dan McKenzie, Mr. Yearsley, Dr. McBride, Dr. Haslam, Dr. Milligan

—— —— of very extensive epithelioma of the fauces, Laryng. 64

——, discussion on case of complete occlusion of posterior nares, Otol. 134

—— —— —— of deaf-mute apparently regaining hearing, Otol. 13

—— —— —— of extensive tuberculosis of larynx, Laryng. 9

—— —— —— of functional aphonia in a male, Laryng. 77

GRANT, J. DUNDAS.—Discussion on case of growth of left vocal cord, Laryng. 96
—— —— on case of laryngeal paralysis, Laryng. 108
—— —— —— of lupus of epiglottis, Laryng. 106
—— —— —— of mucocele of the frontal sinus, Laryng. 71
—— —— —— of nasal obstruction, Laryng. 76
—— —— —— of tuberculous laryngitis treated by thyrotomy, Laryng. 7
—— —— —— of tumour of right thyroid lobe, Laryng. 40
—— —— on clinical pathology of aural discharges, Otol. 121
—— —— on crossed abducens paralysis in a case of cerebellar abscess, Otol. 89
—— —— on extreme form of septal deflection, Laryng. 6
—— —— on fistulous opening in middle line of neck, Laryng. 4
—— —— on mucocele of anterior ethmoidal cell, Laryng. 102
—— —— on a new adenotome, Laryng. 81
—— —— on new instrument for passing suture through epiglottis, Laryng. 96
—— —— on operative surgery of labyrinthitis, Otol. 77
—— —— on purulent otitis media with deep ulceration of external meatus in a case of secondary syphilis, Otol. 88
—— —— on subcutaneous induration of neck of uncertain origin, Laryng. 112
—— —— on subglottic laryngeal tumour, Laryng. 90
—— —— on sudden deafness in case of syphilis, Otol. 8
—— and McKENZIE, D.—Case of median cervical fistula, shown after operation (with microscopic specimen), Laryng. 63; pathological report, W. Wingrave, 63, Dr. D. McKenzie (reply)
Granuloma annulare, cases shown (E. G. Graham Little), Derm. 33, 47
—— ——, histology of, Derm. 146
—— ——, incidence of, Derm. 145
—— ——, nature of, Derm. 148-151
—— ——, nodule initial lesion of, Derm. 142-145
—— ——, rarity of, Derm. 145
—— ——, record of cases reported, Derm. 95-141
—— ——, relation to erythema elevatum diutinum, Derm. 149
—— —— —— to lichen planus, Derm. 149, 150
—— —— —— to lupus erythematosus, Derm. 149
—— —— —— to sarcoid tumours, Derm. 150, 151
—— ——, treatment, Derm. 151
—— ——, tuberculous associations, Derm. 148, 150, 151
——, infective, case of, report on (J. H. Stowers), Derm. 32
—— —— of septic origin (J. H. Stowers), Derm. 7
—— of mouth with penile ulcer (J. H. Sequeira), Derm. 57
Granulomata, vegetating, on face (T. Colcott Fox) Derm. 25
Granulomatous swellings at left angle of mouth and in right inguinal region (J. H. Sequeira), Derm. 92
Grape-like sarcoma of cervix uteri (F. J. McCann), Obst. 253
GRAY, ALBERT A.—Discussion on deafness resulting from epidemic cerebrospinal meningitis, Otol. 87
——, discussion on operative surgery of labyrinthitis, Otol. 77
—— —— on sudden deafness in case of syphilis. Otol. 9
—— —— on vestibulotomy, Otol. 154
——, specimens and photographs of pathological conditions found in the labyrinth, Otol. 93; discussion (p. 93): Mr. Yearsley, Dr. Kelson, Mr. R. Lake, Dr. Gray (reply)
——, stereoscopic photographs illustrating the comparative anatomy of the labyrinth of reptiles, birds and mammals, Otol. 164.
GRAY, A. M. H.—Case of myxœdema with unusual features, Clin. 26; discussion (p. 28): Dr. Parkes Weber, Dr. A. E. Garrod, Sir Thomas Barlow, Dr. Gray (reply)

Green, A. Stanley.—The diagnostic value of the Röntgen rays in some diseases of the chest; an address introductory to a discussion on the subject, Electr. 35; discussion continued by (p. 44) Mr. W. Deane Butcher, Dr. David Arthur, Dr. Halls Dally, Dr. Harrison Orton, Dr. Squire, Dr. Lees, Dr. Lyster, Dr. D. Somerville, Dr. Samuel West, Dr. Hinds Howell, Dr. Allpress Simmons, Dr. J. A. Codd, Dr. Green (reply)

—— and Lack, H. L.—Case of subglottic laryngeal tumour, Laryng. 90; discussion (p. 90): Dr. S. Green, Dr. W. Williams, Dr. Donelan, Dr. Grant, Dr. F. Powell, Dr. H. J. Davis, Dr. D. McKenzie, Mr. C. Fox, Dr. S. Green (reply)

Greenwood, Major, jun.—Discussion on Mendelism in relation to disease, Epid. 162

Griffith, W. S. A.—Discussion on case of difficult labour, Obst. 148

——, discussion on physiological action of placenta, Obst. 12

—— —— on pubiotomy, Obst. 30

—— —— on the supports of the pelvic viscera, Obst. 224

—— —— on unilateral hæmatometra removed by operation, Obst. 41

Grimmer, G. K.—Case of partial occlusion of both anterior nares by a congenital cutaneous web, Laryng. 66; discussion (p. 66): Dr. Barry Ball, Sir F. Semon, Dr. P. McBride, Dr. S. Spicer, Dr. Grimmer (reply)

Grove House Home for Girls, Brislington, near Bristol, outbreak of typhoid fever at, caused by "carrier," Epid. 185

Grünbaum, Otto, and Austin, L. J.—Some experiences with the tuberculin ophthalmic reaction, Path. 74

Guinea-pigs, experiments on, proving non-convertibility of human bacillus into avian, Path. 70, 71

——, inoculation of, with suspensions of tuberculous material from birds, Path. 50

—— —— ——, arrest and retrogression of process, Path. 52

——, limited pathogenicity of avian tubercle bacillus to, Path. 70

——, tuberculous organs of, infected by means of human sputum, inoculation of pigeons by, Path. 31

Guinea-worm, curative drug treatment of, undiscovered, Therap. 26

—— parasitic in man, Therap. 25

——, provision for continuance of species, Therap. 25

Gumma of larynx (L. Jones), Laryng. 19

Gummata (cerebral) some peculiarities of (J. S. Collier), Neur. 15

Gummatous infiltration, diffuse, of left half of nose (W. Jobson Horne), Laryng. 14

—— ulceration, extensive, round mouth (J. H. Sequeira), Derm. 185

—— —— ——, of nose and lip, with gummatous hepatitis (J. H. Sequeira), Derm. 183

Gums, fibromata of, diffuse (A. Evans), Odont. 125

Guthrie, Leonard G.—Arteritis obliterans of right upper extremity, associated with malformation of both clavicles, Neur. 101; discussion (p. 102): Dr. Parkes Weber, Dr. F. Buzzard, Dr. Guthrie (reply)

——, case of muscular atrophy, with delayed thermal sensation (shown by Mr. Kilgour), Neur. 10

——, discussion on cerebral degeneration with changes at the macula, in four members of a family, Neur. 99

—— —— on hereditary spastic paralysis, Neur. 91

—— —— on "tics," Neur. 46

—— —— on tumour of motor area of brain which was operated upon, Neur. 97

——, functional trismus, Neur. 103

——, remarks on rheumatism and chorea in childhood, Therap. 65; discussion (p. 79): Dr. Cecil Wall, Dr. Lees, Dr. Gordon Sharp, Dr. J. Gray Duncanson, Dr. Guthrie (reply)

——, two cases of cerebral diplegia (non-congenital) in children, Neur. 67

—— and Mayou, Stephen.—Right hemiplegia and atrophy of left optic nerve, Clin. 180

Guy's Hospital, statistics of complications of pneumonia at (1902-6), (J. Fawcett, H. C. C. Mann, and H. C. Cameron), Med. 69

Gyrus, post-central, gross lesion of, associated with astereognosis (P. Stewart), Clin. 220

Habershon, S. H.—Discussion on treatment of functional dyspepsia, Therap. 7
Habit tics, Neur. 42
Hadley, W. J.—Discussion on pneumonia and its complications, Med. 27
——, statistics of complications of pneumonia from the London Hospital (1897-1906), Med. 57
Hæmangiectatic hypertrophy of foot (F. Parkes Weber), Clin. 193, Derm. 59
Hæmatoma, subperiosteal, difficulty of diagnosis from periosteal sarcoma, Path. 90, 91
—— (suppurating) of septum nasi, with fistulous opening in the gingivo-labial recess (D. McKenzie), Laryng. 92
Hæmatometra, unilateral, accompanying uterus bicornis duplex (T. Wilson), Obst. 70
—— —— removed by operation (T. Wilson), Obst. 41
Hæmatosalpinx complicating myoma of uterus (H. Macnaughton Jones), Obst. 122
—— with acute symptoms, following retroversion of fibroid uterus (A. Doran), Obst. 174
Hæmophilia, inheritance of, investigation of problem attempted by crossing experiment in sheep, Epid. 156, 157
—— limited practically to male sex, Epid. 153
——, pedigree illustrating inheritance of, Epid. 154
Hæmoptysis in pneumonia, Med. 44
Hæmorrhage in goitre cause of dyspnœa, Surg. 26
——, risk of, in cholecystectomy, Surg. 102, 103
——, treatment by gelatine diet successful, Med. 247
——, uncontrollable, in operations on jaundiced patients, Surg. 102, 103
—— (cerebral) as immediate cause of death in eclampsia of pregnancy, Obst. 92-94
—— —— into pons Varolii as immediate cause of death in eclampsia of pregnancy (N. C. Carver and J. S. Fairbairn), Obst. 79
—— (rectal), with bilateral telangiectases of trunk (T. Colcott Fox), Derm. 66
—— (venous, post-operative), after removal of goitre, Surg. 32
Hæmorrhages, tendency to, in cases of lymphocythæmia, Clin. 159
—— (recurrent, multiple hereditary), developmental telangiectases associated with (F. Parkes Weber), Laryng. 43
Haffkine, W. M., C.I.E.—On the present methods of combating the plague, Epid. 71. [For discussion see under *Thompson*, J. Ashburton—Protection of India from invasion by bubonic plague]
Hairball in stomach; removal (G. F. Still), Clin. 212
Hajek's hook in right sphenoidal sinus, X-ray photograph showing (H. J. Davis), Laryng. 138
Halban and Tandler, views on function of levator ani muscle, quoted, Obst. 213
—— ——, views of, on pelvic visceral supports, quoted, Obst. 204
Hales, Stephen, views on importance of use of enema before giving purgatives, quoted, Med. 146
Hall, F. de Havilland. Case of functional aphonia in a male, Laryng. 77; discussion (p. 77): Sir F. Semon, Mr. H. Tilley, Dr. D. Grant, Dr. H. J. Davis, Dr. Hall (reply)
——, discussion on case of angina pectoris with aortitis, Clin. 118
—— —— —— of functional aphonia, Laryng. 79
—— —— —— of tuberculous laryngitis treated by thyrotomy, Laryng. 7
—— —— —— of tumour of right thyroid lobe, Laryng. 40
—— —— on localized pulsating growth of middle turbinate body, Laryng. 1
—— —— on neoplasm of right tonsil, Laryng. 141
—— —— on pathology and treatment of constipation, Med. 149
—— —— on tuberculosis and syphilis in larynx, Laryng. 136
Hall, I. Walker, and Davies, D. S.—Typhoid carriers, with an account of two institution outbreaks traced to the same "carrier," Epid. 175, 227
Hallager, on the causation of epilepsy quoted, Med. 87
Hamer, W. H.—Discussion on medical inspection in schools, Epid. 67
——, the relation of the *Bacillus typhosus* to typhoid fever, Epid. 204, 227
Hammering, in ununited fractures, Surg. 237

Hand, chronic X-ray dermatitis of; removal of warts by measured doses of X-rays (J. H. Sequeira), Derm. 78

——, hands and feet only affected in case of lupus erythematosus (H. G. Adamson), Derm. 2

——, lupus erythematosus affecting hands, ears, and scalp (J. M. H. MacLeod), Derm. 88

——, œdema of hands and feet with mediastinal affection (F. Parkes Weber), Clin. 192

——, osteo-arthritis of hands, with disseminated lupus (A. Whitfield), Derm. 93

——, ringed eruption on (G. W. Dawson), Derm. 65

——, wasting of small muscles of, in girl aged 20, and her father aged 46 (T. Thompson), Neur. 63

HANDFIELD-JONES, M.—Tumour of fœtal head (shown by Harold S. Sington, introduced by Dr. Handfield-Jones), Obst. 158

HANDLEY, W. SAMPSON.—Case of complete transverse resection of the pharynx with laryngectomy for malignant growth (squamous-celled carcinoma) of the posterior pharyngeal wall, Clin. 66; discussion (p. 68): Mr. Herbert Tilley, Mr. R. D. Jocelyn Swan, Mr. Handley (reply)

——, discussion on case of ascites cured by permanent drainage through femoral ring, Clin. 50

—— —— on cases showing improved phonetic condition—(1) after excision of larynx, (2) after removal of vocal cord, Clin. 185

——, two cases of lymphangioplasty for the brawny arm of breast cancer, Clin. 186; discussion (p. 189): Mr. Cecil Leaf, Dr. Parkes Weber, Dr. zum Busch, Dr. Copeman, Mr. Handley (reply)

HANN, R. G.—Discussion on splenic polycythæmia with cyanosis, Clin. 42

——, polycythæmia with enlarged spleen, without cyanosis, in a girl aged 18, Clin. 59; discussion (p. 60): the President (Sir T. Barlow), Dr. Robert Hutchison, Dr. Parkes Weber

Hare, F., on causation of cardiac inhibition in epilepsy, quoted, Med. 82

—— on epileptic aura, quoted, Med. 94

HARMER, W. D.—Case of pulsating angeioma of the nose, Laryng. 142; discussion (p. 142): Mr. De Santi, Dr. D. McKenzie (reply)

HARRIS, A. BUTLER.—Discussion on complications of pneumonia, Med. 45

HARRIS, W.—Case of paralysis agitans, Neur. 98

——, case of peroneal atrophy, Neur. 54

—— —— of right-sided extra-cerebellar tumour, Neur. 98

—— —— of slowly progressive hemiplegia, Neur. 5; discussion (p. 6): Dr. Parkes Weber, Dr. C. E. Beevor

——, discussion on case of extreme lordosis of two years' standing, Neur. 58

—— —— on cases of myatonia congenita and of toxic affection of muscles and lower neurones, Neur. 4

—— —— on case of nerve-root grafting, Neur. 9

—— —— on tics, Neur. 45

——, two cases of cerebellar ataxy, Neur. 52; discussion (p. 53): Dr. G. Holmes, Dr. W. Harris (reply)

Hart, D. Berry, case of pubiotomy referred to, Obst. 21, 22

HARTIGAN, T. J. P.—Case of erythema, Derm. 14

——, case of leprosy, Derm. 83

——, discussion on ionic medication, Electr. 79

—— —— on mycosis fungoides, Derm. 168

—— —— on unusual case of pustular vegetating dermatitis with pigmentation changes, Derm. 168

——, five cases of lupus erythematosus treated by a new method, Derm. 45

——, multiple rodent ulcer, Derm. 8

Hartley, P. Horton-Smith, persistence of *Bacillus typhosus* in human body as source of reinfection; his conclusions verified, Epid. 175, 176

Harvey, experiments on production of arteriosclerosis quoted, Therap. 34, 35

HASLAM, W. D.—Discussion on unilateral hysterical nerve deafness, Otol. 128

HAULTAIN, F. W. NICOL.—Abdominal hysterotomy for chronic uterine inversion: a record of three cases, Obst. 279; discussion (p. 288): Dr. Champneys, Dr. Heywood Smith, Dr. Amand Routh, Dr. Frank E. Taylor, Dr. Herbert Spencer, Dr. Haultain (reply)
HAWARD, J. WARRINGTON.—Discussion on complete gastrectomy, Surg. 91
—— —— on removal of goitre by operation, Surg. 76
—— —— on sciatica and its surgical treatment, Surg. 174
—— —— on subcoracoid dislocation of humerus, Surg. 197
—— —— on treatment of coxa valga, Surg. 141
—— —— —— of ulcerative proctitis by zinc cataphoresis, Surg. 184
HAWKINS, FRANCIS H.—Case of ascites; paracentesis performed twenty-five times in one year and three months; patient quite well seven years later, Clin. 190; discussion (p. 192): Dr. Parkes Weber, Dr. Hawkins (reply)
——, case of multiple rheumatic nodules in an adult, Clin. 237
HAWKINS, H. P.—Discussion on acute ulcerative colitis, Med. 164
HAWTHORNE, C. O.—Discussion on case of multiple hereditary developmental angiomata, Clin. 66
Hay and Moore, on convulsions and cardiac attacks, quoted, Med. 75
HEAD, HENRY, F.R.S.—Discussion on case of amyotonia congenita, Neur. 62
——, discussion on case of disease of the cervical vertebræ with pressure symptoms, Neur. 13
—— —— on case of nerve-root grafting, Neur. 8
—— —— on cases of wasting of small muscles in hand, Neur. 64
—— —— on hereditary spastic paralysis, Neur. 92
—— —— on intermittent claudication in lower extremity (angina pedis or angina cruris) from chronic arteritis obliterans, Neur. 51
—— —— on mechanism of nystagmus, Neur. 82
—— —— on multiple neuro-fibromata and spastic paraplegia, Neur. 63
—— —— on syringomyelia, Neur. 90
——, opening of discussion on "tics," Neur. 41
Head, unusual position of, in case of bulbar paralysis (J. F. Little), Neur. 70
—— and jaws of adult *Hemirhamphus*, two specimens (A. Hopewell-Smith), Odont. 61
Headache in labyrinthitis, Otol. 56
Hearing apparently being regained by deaf-mute (L. A. Lawrence), Otol. 13
Heart, action of digitalis on, Med. 258-261, 262, 270, 271, 272, 273, 274
——, action of digitalis on (J. Mackenzie), Therap. 29
—— —— —— ——, beneficial during dilatation, Therap. 29,
—— —— —— ——, effect on passage of stimulus for contraction from auricle to ventricle, Therap. 30
—— —— —— ——, inhibitory, Therap. 31
—— —— —— ——, little effect under normal conditions, Therap. 29
—— —— —— ——, non-beneficial in cases of failure without dilatation, Therap. 30
—— —— —— ——, restores function of tonicity, Therap. 30
—— —— —— ——, stimulating, Therap. 31
—— —— of, effect of nervous system on, Med. 271, 272, 274
——, dilatation of, produced by chloroform, Med. 271, 272
—— —— of left ventricle in acute infective diseases, Med. 273, 274
——, disease of, associated with syncopal and convulsive seizures, Med. 73
—— ——, congenital, in adults, cases (R. Murray Leslie), Clin. 34
—— —— consequent on rheumatism, slowing of pulse-rate under digitalis administration, Therap. 30
—— —— percentage in cases of chorea, Therap. 74
—— —— preceding acute suffocative pulmonary œdema, Med. 154
—— —— ruptured aortic valve (Dr. S. Taylor), Clin. 97
——, muscle of, see *Muscle*, cardiac
——, percussion of, importance, Med. 273
——, post-mortem condition in cases of lymphocythæmia, Clin. 165

Heart, tetanization of, Med. 254
Heart-block, see *Stokes-Adams* disease
Heart-failure in pneumonia, myasthenic and mechanical, Med. 39, 40
Heat, local application of, in treatment of gastric motor spasm, Therap. 6
Heath, P. Maynard.—Caries of spine with clubbing of fingers and toes, Clin. 234
Hedley, J. P.—Bilateral hæmorrhagic ovarian cysts, Obst. 96; discussion (p. 98): Dr. Mansell Moullin, Dr. Lewers
—, hydrosalpinx with torsion of pedicle, Obst. 95
Hemianæsthesia in case of unilateral hysterical nerve deafness (J. Dundas Grant), Otol. 127
Hemicrania, association with epilepsy, Med. 89
Hemihypertrophy (J. P. Lockhart Mummery), Clin. 61
Hemiplegia, alternate contraction of opposing muscles in, Neur. 73
——, slowly progressive (W. Harris), Neur. 5
—— with involuntary movements (C. Thomson), Neur. 55, 95
—— (right), with atrophy of left optic nerve, Clin. 180
—— ——, following operation (G. Ogilvie), Neur. 94
Hemirhamphus, adult, head and jaws of, two specimens (A. Hopewell-Smith), Odont. 61
Henry, Martin.—Discussion on treatment of children from dental aspect, Odont. 73
Hens, observations on, respecting relation of egg-laying to blood-calcium, Obst. 295, 296
Hepatitis, gummatous, in extensive gummatous ulceration of nose and lip (J. H. Sequeira), Derm. 183
Hepato-splenomegaly, case with ascites (H. Batty Shaw), Clin. 56
Heredity, relation to dental disease (S. Mummery), Odont. 108
——, Weismann's theory of, diagrammatic explanation, Odont. 111
—— (Mendelian theory of), see *Mendelism*
Herman, G. Ernest.—Discussion on case of difficult labour, Obst. 149
——, discussion on myomectomy during pregnancy, Obst. 136
——, his method of symphysiotomy, quoted, Obst. 20
Hermaphroditic condition of sexual glands of birds, Path. 7
Hermaphroditism, uterus occupying sac of inguinal hernia in case of (T. H. Kellock), Clin. 111
——, glandular, incomplete, in domestic fowl (S. G. Shattock and C. G. Seligmann), Path. 3
Hern, William.—Discussion on dental uses of paraform, Odont. 43
——, discussion on treatment of children from dental aspect, Odont. 68
—— —— on treatment of pyorrhœa, Odont. 67
Hernia (inguinal), sac of, occupied by uterus in case of hermaphroditism (T. H. Kellock), Clin. 111
—— —— —— —— —— —— ——, operation, cure, Clin. 112
Herpes complicating pneumonia, Med. 14
Herpetiform eruption, feigned (H. G. Adamson), Derm. 82
Herringham, W. P.—Case of pigmentation with enlarged spleen and leucopenia, ? splenic anæmia, Clin. 33
——, discussion on acute ulcerative colitis, Med. 163
—— —— on complications of pneumonia, Med. 20
—— —— on treatment of pneumothorax with aspiration under X-rays, Clin. 40
——, neuritis of brachial plexus, probably arising from arthritis of shoulder-joint, Clin. 31; discussion (p. 32): Dr. Buzzard, Dr. Sidney Phillips, Mr. Jonathan Hutchinson, jun., Dr. Herringham (reply)
Herschell, George.—Discussion on treatment of functional dyspepsia, Therap. 8
Hertford Epidemic (1904-5), Epid. 112
Hertz, A. F.—The pathology and treatment of chronic constipation, Med. 119; discussion (p. 148): Dr. Morley Fletcher, Dr. C. E. Beevor, Dr. de Havilland Hall, Dr. Hertz (reply)
Heterozygote, definition of, Epid, 138
Hiccough accompanying acute ulcerative colitis, Med. 161, 163
High frequency currents, treatment by, Electr. 9
—— —— —— in treatment of atonic conditions of digestive system, Electr. 110

Highmore, antrum of, see *Antrum*, maxillary
Hill, Leonard, on production of epileptic aura, quoted, Med. 95
HILL, WILLIAM.—Case of tuberculosis of the larynx, Laryng. 64
——, discussion on case for diagnosis, Otol. 28
—— —— on case of chronic empyema of left frontal sinus, treated by simplified method of Killian's operation, Laryng. 36
—— —— on case of laryngeal disease, Laryng. 12
—— —— —— of tuberculous laryngitis treated by thyrotomy, Laryng. 8
—— —— —— of ulceration and destruction of epiglottis, Laryng. 34
—— —— on impaction of bone in larynx, Laryng. 5
—— —— on proptosis due to distension of frontal sinus, Laryng. 50
—— —— on pulsating growth in left external auditory meatus, Otol. 33
—— —— on specimen of large post-nasal polypus removed from boy aged 14½, Laryng. 122
Hip-joint, dislocation of, congenital, associated with coxa valga, Surg. 121
—— —— —— in child aged 2 weeks, with multiple dislocations, result of malposition *in utero* (T. H. Openshaw), Clin. 106
——, tuberculous disease of, treatment by oral administration of tuberculin and horse serum, Med. 207
—— (left), dislocation, traumatic, replaced by manipulation thirteen months later (T. H. Openshaw), Clin. 43
Hippocrates' views on menstruation quoted, Obst. 291
HODGSON, R. H.—Discussion on cerebral hæmorrhage in eclampsia of pregnancy, Obst. 94
——, discussion on ovarian dermoid with secondary cysts connected with omentum, Obst. 118
—— —— on vaccine treatment by the month, Med. 249
Hog cholera bacillus, infective capabilities of, Epid. 216, 221
HOLLAND, C. THURSTAN.—Discussion on X-ray diagnosis of renal and ureteral calculi, Electr. 31
HOLLAND, EARDLEY.—Melanotic tumour of the vulva, Obst. 124
—— and WILLIAMSON, HERBERT.—Case of intra-uterine death of the fœtus occurring in six consecutive pregnancies, with observations upon the importance of the examination of the fœtal tissues for the presence of the *Spirochæta pallida*, Obst. 161; discussion (p. 170): Dr. Herbert Spencer, Dr. Amand Routh, Dr. Maxwell, Dr. Beckett Overy
HOLMES, GORDON.—Discussion on cases of cerebellar ataxy, Neur. 53
——, discussion on cases of myatonia congenita and of toxic affection of muscular and lower neurones, Neur. 4, 5
—— —— on case of tremor of the right arm associated with epilepsy, Neur. 56
—— —— on cerebral degeneration, with changes at the macula in four members of a family, Neur. 99
—— —— on hemiplegia with involuntary movements, Neur. 56
—— —— on tumour of motor area of brain which was operated upon, Neur. 97
—— and BATTEN, F. E.—The nervous system of a dog which suffered from ataxia and involuntary movements, Neur. 22; discussion (p. 26): Dr. C. E. Beevor, Dr. Ferrier, Dr. Purves Stewart, Sir Victor Horsley
Homalomyia, larvæ of various species of, passed by intestine, Path. 150
Homozygote, definition of, Epid. 138
Hood, Donald, on etiology of rötheln, alluded to, Epid. 108
HOPEWELL-SMITH, A.—Discussion on termination of nerves in teeth of mammalia, Odont. 57
——, (1) two specimens of the head and jaws of the adult *Hemirhamphus*; (2) a specimen showing developmental defects occurring in the upper jaw of a pike (*Esox lucius*) Odont. 61
HOPSON, MONTAGU.—Discussion on difficult extraction, Odont. 138
HORNE, W. JOBSON.—Case of diffuse gummatous infiltration of the left half of the nose, Laryng. 14
——, case of lipoma or cystoma of the neck, Laryng. 38; discussion (p. 38): Dr. H. J. Davis, Dr. Donelan, Mr. Clayton Fox

Horne, W. Jobson.—Case of lupus of the epiglottis, Laryng. 106; discussion (p. 106): Dr. McBride, Dr. J. B. Ball, Dr. Grant, Dr. StClair Thomson, Dr. Jobson Horne (reply)
——, case of symmetrical neoplasms on the vocal cords, Laryng. 13
—— —— of ulceration and destruction of the epiglottis, Laryng. 33; discussion (p. 34): Dr. H. J. Davis, Mr. Cresswell Baber, Mr. Barwell, Sir Felix Semon, Dr. FitzGerald Powell, Mr. Waggett, Dr. William Hill, Dr. Jobson Horne (reply)
—— —— of unilateral paralysis of the tongue, Laryng. 53; discussion (p. 54): Dr. Davis, Mr. Steward, Dr. Dan McKenzie, Mr. Clayton Fox, Dr. Jobson Horne (reply)
——, discussion on atresia of naso-pharynx, Laryng. 16
—— —— on bilateral sarcoma of upper maxillæ, Laryng. 21
—— —— on bleeding polypus of the septum, Laryng. 28
—— —— on bony outgrowths from maxilla and mandible, Laryng. 95
—— —— on cases after radical maxillary antrum operation, exemplifying simplification of the after-treatment, Laryng. 59
—— —— on case for diagnosis, Otol. 27
—— —— —— of girl aged 17, with a genuine baritone voice, Laryng. 61
—— —— —— of laryngeal disease, Laryng. 120
—— —— —— of tuberculous laryngitis treated by thyrotomy, Laryng. 8
—— —— —— of septal deflection in a phthisical patient, Laryng. 112
—— —— —— showing remarkable development of orbito-ethmoidal cells, Laryng. 121
—— —— clinical pathology of aural discharges, Otol. 118
—— —— on impaction of bone in larynx, Laryng. 5
—— —— on keloid following mastoid operation, Otol. 22
—— —— on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 105
——, exhibition of autograph letter by Johann Nepomuk Czermak (1828-73), Laryng. 83
——, preparations illustrating diseases of the trachea, Laryng. 37; discussion (p. 38): Dr. H. J. Davis, Mr. Herbert Tilley, Dr. Jobson Horne (reply)
——, specimens from cases of sarcoma and cases simulating sarcoma in the upper air passages, Laryng. 100
—— —— showing the pathogenesis of pachydermia laryngis verrucosa et diffusa, Laryng. 132
Horns, inheritance of, in sheep, experiments made to investigate, Epid. 154, 155
Horrocks, Major.—Discussion on etiology and epidemiology of typhoid fever, Epid. 224
Horrocks, Peter.—Discussion on unilateral hæmatometra removed by operation, Obst. 41
——, tubercle of cervix uteri, Obst. 66; discussion (p. 67): Dr. Herbert Spencer, Sir Arthur Macan, Dr. Horrocks (reply)
Horse, molar tooth of, effects of chronic suppuration in (J. G. Turner), Odont. 26
Horse ringworm, pustular, in child ("conglomerate folliculitis"), Derm. 63
Horse serum, anti-autolytic powers of, Med. 246
—— ——, antitryptic power of, Med. 242
—— ——, injection in treatment of pneumonia, Med. 67
—— ——, local administration, Med. 242
—— —— —— —— —— value, Med. 246
—— —— (fresh), hypodermic administration increases coagulability of blood, Med. 198
—— —— ——, oral administration of, effects of, Med. 232
—— —— —— —— —— better when given with tuberculin, Med. 235, 237
—— —— —— —— in broncho-pneumonia, Med. 197
—— —— —— —— ——, value of, estimated, Med. 242
—— —— —— with tuberculin (T.R.) in caseating pulmonary tuberculosis (A. Latham), Clin. 100
—— —— —— —— —— —— and other vaccines (A. Latham, H. D. Spitta and A. C. Inman), Med. 195-239
—— —— ——, use as dressing in mastoid operations, Med. 248
—— —— ——, see also *Pneumococci*, *Staphylococci*, *Streptococci*, *Tuberculin* (T.R.)
Horsford, C. A.—Discussion on case of functional aphonia, Laryng. 79
——, discussion on extensive cellulitis of right side of neck, Laryng. 11

HORSFORD, C. A.—Discussion on paresis of right vocal cord, Laryng. 3
——, girl aged 17, with a genuine baritone voice, Laryng. 61; discussion (p. 61): Dr. Donelan, Dr. Jobson Horne, Dr. Aikin, Sir F. Semon, Mr. Horsford (reply)
——, ? new instrument devised to facilitate intralaryngeal operations, Laryng. 24; discussion (p. 24): Mr. Stuart-Low, Mr. De Santi, Dr. StClair Thomson, Sir Felix Semon, Mr. Waggett, Mr. Horsford (reply)
——, new instrument for passing a suture through the epiglottis, Laryng. 95; discussion (p. 96): Dr. D. Grant, Dr. H. J. Davis, Dr. S. Spicer, Mr. Stuart-Low, Dr. F. Powell Mr. Horsford (reply)
——, patient in whom a small fibroma had been removed from the left vocal cord, Laryng. 24
HORSLEY, Sir VICTOR.—Discussion on cerebral gummata, Neur. 19
——, discussion on case of tubercle of one sixth nucleus, Neur. 40
—— —— on condition of nervous system of ataxic dog, Neur. 26
—— —— on removal of goitre by operation, Surg. 76
—— —— on right hemiplegia following operation, Neur. 95
HORT, E. C.—Discussion on vaccine treatment by the mouth, Med. 241
Horton-Smith, see *Hartley*, P. Horton-Smith
HOSFORD, J. STROUD, and PARKINSON, J. PORTER.—Cerebellar tumour with proptosis, Clin. 124; discussion (p. 127): Mr. Hosford, Sir T. Barlow, Sir Dyce Duckworth, Dr. Porter Parkinson (reply)
House-incidence theory of transmission of typhoid fever untenable, Epid. 205, 206
HOVELL, T. MARK.—Discussion on functional aphonia, Laryng. 80
HOWARD, RUSSELL.—A case of congenital defect of the muscular system (dystrophia muscularis congenita) and its association with congenital talipes equino-varus, Path. 157
HOWELL, C. M. HINDS.—Discussion on case of nerve-root grafting, Neur. 8
——, discussion on diagnostic value of Röntgen rays in some diseases of the chest, Electr. 52
—— —— on treatment of leukæmia, exophthalmic goitre and malignant disease by X-rays, Electr. 61
—— —— on wasting of the small muscles of the hand in a girl aged 20, and her father aged 46, Neur. 64
——, two cases of tabes, Neur. 65; discussion (p. 66): Dr. Beevor
Huber, J. C., case of passage of larvæ of *Eristalis* through intestine reported by him, Path. 151
Human agency unimportant in spread of plague, Epid. 71, 72
HUME, F. N.—Discussion on rubella, Epid. 128
Humerus, dislocation of, dissection from behind, Surg. 190, 191
—— —— —— dissection from in front, Surg. 188, 189
—— —— —— unreduced, Surg. 198
—— —— —— (subcoracoid, old), exposure from behind, Surg. 193
—— —— —— ——, reduction by excavating glenoid cavity through posterior intermuscular incision (W. G. Spencer), Surg. 187
—— —— —— —— —— —— —— —— —— —— ——, advantages, Surg. 197
——, fracture of, sarcoma arising at site of, Path. 100
—— —— (compound) (Rushton Parker), Surg. 221
—— —— ——, report of cases, Surg. 221, 231
—— —— ——, treatment, Surg. 221-231
—— —— —— ——, application of splints, Surg. 230, 231
—— —— —— ——, slinging of arm to neck and flexion of elbow to acute angle, Surg. 222-225, 230
—— —— ——, old, with osteo-arthritis of elbow (R. J. Godlee), Clin. 95
—— —— —— (ununited), cases of (Rushton Parker), Surg. 232
—— —— ——, treatment by planting small fragments of bone between ends (Rushton Parker), Surg. 232
——, muscles behind, normal position, with outstretched arm, Surg. 190, 192
HUMPHREYS, JOHN.—The teeth of fossil fishes, Odont. 7; discussion (p. 15): Mr. D. Gabell, Mr. F. J. Bennett, Mr. H. Baldwin, Mr. Humphreys (reply)

Huntington's chorea, case of (E. F. Buzzard), Neur. 12

HUTCHINSON, JONATHAN, jun.—Case of fusiform aneurysm of the right common carotid artery, Clin. 17, 21

——, discussion on case of recurrent dislocation of shoulders, Clin. 30

—— —— on neuritis of brachial plexus, Clin. 32

——, trigeminal neuralgia: excision of the Gasserian ganglion after unsuccessful intracranial neurotomy of the second and third divisions of the fifth nerve, Clin. 21; discussion (p. 22): Mr. Charles A. Ballance, Mr. Hutchinson, jun. (reply)

HUTCHISON, ARTHUR.—Bilateral sarcoma of the upper maxillæ (with microscopical section), Laryng. 21; discussion (p. 21): Mr. De Santi, Dr. Jobson Horne, Dr. L. H. Pegler, Dr. Scanes Spicer, Dr. Westmacott, Dr. FitzGerald Powell, Dr. H. Smurthwaite, Mr. A. L. Whitehead, Mr. Hutchison (reply)

—— —— —— (report of Morbid Growths Committee on), Laryng. 43

HUTCHISON, ROBERT, allusions to lymphocythæmia in Goulstonian Lectures quoted, Clin. 131

——, discussion on case of enlarged spleen with polycythæmia, without cyanosis, Clin. 60

—— —— on treatment of functional dyspepsia, Therap. 3, 12

Hydatid cyst represented in cases of goitre removed by operation, Surg. 25

—— ——, (?) tumour of mediastinum (H. French), Clin. 93

Hydrochloric acid in treatment of dyspepsia, Therap. 6

—— —— —— —— combined with pepsin, Therap. 8

Hydrosalpinx with torsion of pedicle (J. P. Hedley), Obst. 95

——, large, with small ovarian cyst simulating malignant disease (J. D. Malcolm), Obst. 241

Hyoid bone, fistulous opening in middle line of neck just above (D. McKenzie), Laryng. 3

—— and fibula affected by leontiasis ossea, Path. 93

Hyperacidity, association of gastric hyperæsthesia with, Therap. 7

—— in gastric secretion, Therap. 5

Hyperæsthesia, gastric, association with hyperacidity, Therap. 7

Hyperchlorhydria, treatment of, Therap. 5

—— —— by diet rich in fat, Therap. 11

Hyperidrosis, treatment by hypnotism, Therap. 58

Hyperplasia, unusual degree present in case of chronic eczema of both auricles and meatuses (E. Furniss Potter), Otol. 132

——, subglottic, specimen with considerable narrowing of tracheal airway (H. Tilley), Laryng. 123

Hyperpyrexia, rheumatic, accompanied by acute pulmonary œdema, Med. 155

Hypertrophy, hæmangiectatic, of foot (F. Parkes Weber), Derm. 59

—— —— ——, possibly of spinal origin (F. P. Weber), Neur. 49

Hypnotism, development of patient's will-power under, essential, Therap. 60

——, effect on dogs, Therap. 61, 62

——, England, not France, the birthplace of, Therap. 53

——, extraction of teeth under, Therap. 57

——, induction of, method, Therap. 59

—— in treatment of hyperidrosis, Therap. 58

—— —— —— of hystero-epilepsy, Therap. 56

—— —— —— of sciatica, Therap. 54

—— —— —— of urinary incontinence, Therap. 56

—— not an universal remedy, Therap. 60

——, operations under, Therap. 57

——, origin and development of (J. Milne Bramwell), Therap. 49

—— —— of scientific study of, Therap. 51, 52

——, phenomena of, dependent on condition of patient, Therap. 51, 52

——, term substituted for that of "mesmerism" by Braid, Therap. 52

——, value of, in comparison with other forms of treatment, Therap. 60

—— ——, in functional nervous disorders, Therap. 60

Hypnotism, voluntary compliance an essential of, Therap. 52
——, see also *Anæsthesia*, hypnotic
—— —— *Mesmerism*
Hypochlorhydria, treatment of, Therap. 6
Hysterectomy (subtotal) at fourth month in case of pregnancy complicated by large cervical fibroid (J. Bland-Sutton) Obst. 17
—— —— in treatment of fibroid tumours complicating pregnancy, Obst. 317, 319
—— (supravaginal) with myomectomy in case of pregnancy, complicated with large retroperitoneal and subligamentous fibromyoma (J. H. Dauber) Obst. 132
—— (vaginal) after removal of grape-like sarcoma of cervix uteri (F. J. McCann), Obst. 253
—— (Wertheim's method) for advanced carcinoma of cervix (C. H. Roberts), Obst. 61; (A. H. N. Lewers), Obst. 100
—— —— —— —— —— ——, value of, Obst. 101
——, see also *Cæsarean hysterectomy*
—— —— *Panhysterectomy*
Hysteria, manifestations of, differential diagnosis from tics, Neur. 44
—— provoked by sensational experiments in hypnotism, Therap. 64
—— with unusual laryngeal manifestations (Sir F. Semon), Laryng. 133
——, see also *Nerve deafness*, hysterical
Hystero-epilepsy, treatment by hypnotism, Therap. 56
Hysterotomy (abdominal) in treatment of chronic inversion of uterus (F. W. N. Haultain), Obst. 279, 283, 286

Icebag in treatment of rheumatic pericarditis, Therap. 85
Idio-muscle reflex, Neur. 72
Ilio-coccygeus muscle, anatomy and functions, Obst. 208, 210
—— —— ——, value of, Obst. 202
Immigrants (unvaccinated) from Venezuela to Trinidad, danger from, Epid. 236
Immunity against effects of pneumococcus, Med. 4
—— conferred by absorption of dead bacilli, Med. 195, 196
—— to disease in wheat, experiments respecting, Epid. 140
——, acquired, Odont. 112
——, natural, Odont. 112
—— (regional) from typhoid fever, Epid. 206-209
Ince, Dr.—Discussion on therapeutics of indigestion, Therap. 21
Incubation period of rubella, Epid. 103
India, encouragement given to mesmerism in, Therap. 50, 51
——, good results of inoculation against plague in, Epid. 79
——, protection of, from invasion by plague, Epid. 81
Indigestion, see *Dyspepsia*
Infant, ectropion of cervical mucosa in (R. Jamison), Obst. 323
Infants, convulsions in, connection with epilepsy, Med. 91
——, Gaertner infection (*Bacillus enteritidis*) in (F. E. Batten and J. G. Forbes), Clin. 81
Infectious diseases, evolution in, Epid. 131
—— ——, part taken by leucocytes in (S. G. Shattock and L. S. Dudgeon), Med. 169-194
—— ——, second attacks in school children, Epid. 59
Infective diseases, addition of melanin to blood from subject of; result, Med. 173, 177
—— —— (blood-cells from cases of), comparative amounts of phagocytosis of melanin on presentation of melanin to washed cells of patients and to washed normal cells in immune serum, Med. 184-191
—— (blood-cells from cases of), production, *in vitro*, of opsonin by patient's cells in patient's serum, Med. 191-194
Infectivity of rubella, Epid. 105, 119, 127
Influenza, dilatation of left ventricle in, Med. 273
Ingluvin, value of, in irritable dyspepsia, Therap. 18, 21

Inguinal region (right), granulomatous swellings at left angle of mouth and in right inguinal region (J. H. Sequeira), Derm. 92
Inman, A. C.—See *Latham*, A. preliminary communication on the administration of tuberculin (T.R.) and other vaccines by the mouth
Inoculation as a means of prevention of plague, Epid. 78, 90, 91
—— —— —— ——, good results in India, Epid. 79
—— treatment of pneumonia by, Med. 31
Inouye and Saiki on urine of epileptics, referred to, Path. 132
Insanity, increase in, due to malnutrition, Odont. 119, 123
——, prevalence of, due to removal of influence of natural selection, Odont. 113
Insecticides, suggested use of, in preventing transmission of plague by fleas, Epid. 91
Instrument, new, for passing a suture through epiglottis (C. Horsford), Laryng. 95
—— ? new, to facilitate laryngeal operations (C. Horsford), Laryng. 24
Interrupted currents, control of, by clockwork resistances, Electr. 115
—— —— for electrical testing and treatment (H. Lewis Jones), Electr. 113
—— ——, necessity for, in electro-therapeutics, Electr. 114
Interrupter of Leduc, Electr. 116, 117
Intestine, larvæ of *Eristalis tenax* passed by (S. G. Shattock), Path. 147
—— —— —— of *Homalomyia* (various species) passed by, Path. 150
——, transference of charcoal through intact intestinal mucosa of, in rabbit, Path. 42
—— (small), one foot in length, adhering to ovarian tumour, both successfully removed (H. R. Spencer), Obst. 172
Intra-abdominal pressure as means of support of pelvic viscera, Obst. 205
—— ——, factors of, Obst. 205, 206
Intracranial pain, source of, Med. 89
—— tumour (H. C. Thomson), Neur. 96
Intralaryngeal operations, ? new instrument to facilitate (C. Horsford), Laryng. 24
Intranasal hypertrophy with sweating of nose (J. Donelan), Laryng. 47
Intra-uterine pressure probable factor in production of congenital absence of pectoral muscles, Clin. 73
Introitus, atresia of, in adult, acquired (H. Macnaughton Jones), Obst. 325
Iodine, application in ionic medication of pelvic disease, Electr. 137
—— and lithium ions in treatment of hydrops articuli of knee, Electr. 81
Ionic medication, Electr. 10
—— ——, choice of drugs for, Electr. 70, 79, 81
—— ——, indications for, Electr. 72
—— —— in treatment of bacteriuria, Electr. 135
—— —— —— of cervicitis, Electr. 134
—— —— —— of diseased roots of teeth in pyorrhœa, Odont. 63, 65, 67
—— —— —— of dysmenorrhœa, Electr. 135
—— —— —— of lupus erythematosus in five cases (T. J. P. Hartigan), Derm. 45
—— —— —— of menorrhagia, Electr. 134, 135
—— —— —— of pelvic disease in women in mild cases, Electr, 136
—— —— —— —— —— —— in obstinate cases (S. Sloan), Electr. 126-138
—— —— —— of septic discharge from vagina, Electr. 133
—— ——, principles of, Electr. 127
—— —— —— (H. Lewis Jones), Electr. 65
—— ——, question of idiosyncrasies of patients, Electr. 77
—— ——, see also *Aniline ions*, *Cocaine ions*, *Copper ions*, *Iodine and lithium ions*, *Magnesium ions*, *Salicylic acid ions*, *Zinc ions*
Ionic solutions, strength of, Electr. 71
Ions, definition of, Electr. 67
——, depth of penetration, Electr. 69, 71
——, medication by, Electr. 10
—— ——, see also *Ionic medication*

Ions, method of determination of quantities set in motion, Electr. 68

——, migration of, Electr. 127, 128, 130, 131

—— ——, demonstration of method, Electr. 65, 66

—— ——, variation in direction of, Electr. 71

Ireland, effect of repeal of Corn Laws in, Epid. 14

——, emigration from, Epid. 10

——, phthisis in, Epid. 36

——, Poor-law administration in, Epid. 18

——, Potato Famine in (1847), Epid. 8

——, poverty and disease in, as illustrated by course of typhus fever and phthisis in (A. Newsholme), Epid. 1

——, residual population of, Epid. 29

——, social and sanitary conditions of, Epid. 22

——, typhus fever in, Epid. 5

—— —— ——, contrasted with phthisis, Epid. 20

——, vagrancy and mendicancy in, after 1847, Epid. 14

Ischæmic disturbance of right upper extremity (F. Parkes Weber and E. Michels), Neur. 100

Ischio-coccygeus muscle, see *Coccygeus muscle*

Isthmian Canal Commission, creation of, Epid. 305

—— —— ——, crusade against land crab, Epid. 313

—— —— ——, preventive measures against malaria, Epid. 312

—— —— —— —— —— —— pneumonia, Epid. 310

—— —— —— —— —— —— yellow fever, Epid. 311

—— —— ——, rigid enforcement of sanitary ordinances, Epid. 309

—— —— ——, sanitary works undertaken by, Epid. 306, 308

Jackson, Dr., on rubella and measles, alluded to, Epid. 108

Jacksonian convulsions, Med. 99

James, W. W.—Notes on a case of extensive necrosis of the mandible, Odont. 29; discussion (p. 31): Mr. F. Coleman, Mr. J. H. Badcock, Mr. W. B. Paterson, Mr. W. W. James (reply)

Jamison, Reginald.—Longitudinal section of the cervix of an infant showing ectropion of the cervical mucosa, Obst. 323

Jaundice, operations on patients affected with, liable to be attended with uncontrollable hæmorrhage, Surg. 102, 103

Jaw, development of, Odont. 19

——, growth of (J. T. Carter), Odont. 17

—— ——, closely related to development of antrum, Odont. 20, 21

—— ——, influence on cranial form, Odont. 23

—— (lower), endothelioma of, treatment by X-rays (W. Ironside Bruce), Electr. 59

—— (upper), growth of, influencing growth of mandible, Odont. 23

—— ——, of pike (*Esox lucius*), specimen showing developmental defects in (A. Hopewell-Smith), Odont. 61

—— ——, and nasal septum, effects of long-neglected adenoids on development of (J. Donelan), Laryng. 84

Jaws and head of adult *Hemirhamphus*, two specimens (A. Hopewell-Smith), Odont. 61

Jensen's tumour, Path. 171; see also *Mouse tumours*

Johnson, Raymond.—Discussion on congenital absence of pectoral muscles, Clin. 74

Johnston-Lavis, H. J.—Discussion on vaccine treatment by the mouth, Med. 246

Joints, post-mortem condition of, in case of congenital defect of muscular system, Path. 161

Jones, D. W. C.—Case of intracranial tumour, Neur. 103; discussion (p. 104): Dr. E. F. Buzzard, Dr. Jones (reply)

Jones, Ernest.—Case of isolated paresis of right serratus magnus (under the care of Mr. A. H. Tubby), Neur. 9; discussion (p. 10): Dr. C. E. Beevor

——, case of primary spastic paraplegia illustrating the nature of Schäfer's reflex, Neur. 59

Jones, Ernest.—Discussion on case of amyotonia congenita, Clin. 49
——, discussion on case of tremor of the right arm, associated with epilepsy, Neur. 56
—— —— on hemiplegia with involuntary movements, Neur. 56
Jones, H. B.—Case of swelling of the tonsil, Laryng. 143
Jones, H. E.—Discussion on lateral sinus thrombosis, Otol. 140
Jones, H. Lewis.—Discussion on electrical treatment of atonic conditions of digestive system, Electr. 107
——, discussion on President's survey of electric therapeutics, Electr. 13
—— —— on treatment of leukæmia, exophthalmic goitre and malignant disease by X-rays, Electr. 61
—— —— on X-ray diagnosis of renal and ureteral calculi, Electr. 34
——, interrupted currents for electrical testing and treatment, Electr. 113
——, principles of ionic medication, Electr. 65; discussion (pp. 76-82): Dr. Lewis Jones, Mr. Deane Butcher, Dr. W. Ironside Bruce, Mr. Frederick C. Wallis, Dr. E. S. Worrall, Dr. Donald Baynes, Dr. Bolton, Dr. J. F. Halls Dally, Dr. E. G. Graham Little, Dr. A. H. Pirie, Mr. T. J. P. Hartigan, Dr. G. B. Batten, Dr. Lewis Jones (reply)
——, skiagram of peculiar injury in the hip region, with perforation of the acetabulum by the head of the femur, Electr. 34
Jones, Lawrence.—Case of gumma of the larynx, Laryng. 19; discussion (p. 20): Mr. Barwell, Dr. Bronner, Mr. Stuart-Low, Mr. Lawrence Jones (reply)
Josué, experiments of, on production of arterial disease in animals quoted, Therap. 33
Jousset and Braillon, on the causation of endocarditis by tubercle bacilli, quoted, Path. 122

Kala-azar, resemblance to acute ulcerative colitis, Med. 165
Kay, Dr.—Case for diagnosis (shown by Sir Malcolm Morris), Derm. 52
Kayser, views on typhoid "carriers" quoted, Epid. 210
Keith, Professor Arthur.—Discussion on the supports of the pelvic viscera, Obst. 216
——, views on expansion of maxillary sinus quoted, Odont. 20
—— —— on intra-abdominal pressure quoted, Obst. 206
—— ——, on the pelvic fascia quoted, Obst. 52
Kellock, Thomas H.—Case of hermaphroditism in which the uterus occupied the sac of an inguinal hernia, Clin. 111; discussion (p. 113): Mr. Pearce Gould, Mr. Kellock (reply)
——, discussion on subcoracoid dislocation of humerus, Surg. 199
Kelly, A. Brown.—Sketches of three patients with multiple telangiectases of the skin and mucous membranes of the nose and throat, Laryng. 44; discussion (p. 46): Sir Felix Semon, Dr. Lambert Lack
Keloid following operation for acute mastoid suppuration (H. Tilley), Otol. 20
Kelson, W. H.—Case of bleeding polypus of the nose, Laryng. 33
——, case of swelling of the right arytænoid with loss of movement of the right vocal cord, Laryng. 108; discussion (p. 108): Dr. StClair Thomson, Mr. Barwell, Dr. FitzGerald Powell, Dr. Kelson (reply)
—— —— of ulceration of the nose, Laryng. 63
—— —— showing bony outgrowths from the maxilla and mandible, Laryng. 94; discussion (p. 94): Mr. H. B. Robinson, Dr. McKenzie, Dr. Westmacott, Dr. Jobson Horne, Dr. Pegler, Dr. Kelson (reply)
——, discussion on case of chronic eczema of both auricles and meatuses, Otol. 133
—— —— —— for diagnosis, Otol. 27
—— —— on clinical pathology of aural discharges, Otol. 121
—— —— on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 104
—— —— on mucocele of anterior ethmoidal cell, Laryng. 102
—— —— on operative surgery of labyrinthitis, Otol. 79
—— —— on specimens and photographs of pathological conditions found in the labyrinth, Otol. 93
—— —— on tumour removed from naso-pharynx, Laryng. 10
——, stenosis of right auditory meatus, Otol. 14; discussion (p. 14): Mr. Whitehead, Mr. Lawrence, Mr. Cheatle, Dr. Urban Pritchard, Dr. Kelson (reply)

Ker, Claude B.—Discussion on rubella, Epid. 129

Keratosis, case for diagnosis (W. T. Freeman), Derm. 14

—— follicularis, advanced, associated with baldness (J. M. H. MacLeod and E. Treacher Collins), Derm. 27

Kerr, J. M. Munro.—Case of ovarian pregnancy associated with an intra-uterine pregnancy, Obst. 268; discussion (p. 277): Dr. Herbert Spencer, Dr. Eden, Dr. Blacker, Dr. Teacher (reply)

Kettle, Mr.—Exhibition of sections and film preparations showing spirochætæ, Derm. 80

Kidney, palpation of, in cases of lymphocythæmia, Clin. 160

——, post-mortem condition in cases of lymphocythæmia, Clin. 167

——, see also *Calculus* (renal)

Kilgour, Mr.—Case of muscular atrophy with delayed thermal sensation (shown for Dr. Leonard Guthrie), Neur. 10

Killian's operation for chronic suppuration in frontal sinus (J. Dundas Grant), Laryng. 113

—— —— (complete) in case of chronic suppurative pansinusitis performed on right side, and on anterior and inferior walls only on left side (H. Tilley), Laryng. 97

—— —— (complete and incomplete) in treatment of cases of frontal sinus disease (W. S. Syme), Laryng. 99

—— —— (simplified method); case of chronic empyema of left frontal sinus exhibited two years after treatment by (H. Tilley), Laryng. 36

Kinematograph, use of, in teaching and recording cases of nervous diseases (H. Campbell Thomson), Med. 150

King's College Hospital, statistics of complications of pneumonia at (1897-1906) (J. C. Briscoe and H. W. Wiltshire), Med. 60

Klang (Federated Malay States), suppression of malaria at, Epid. 315

Knee-jerk present in pneumonia, Med. 36, 41, 42

Knee-joint, dropsy of, treatment by iodine and lithium ions, Electr. 81

——, osteo-arthritis of, with old-standing dislocation of patella (R. J. Godlee), Clin. 96

——, tuberculous synovitis of, arthrectomy twice performed (D. Drew), Clin. 10

Koch, Robert, statement respecting administration of tuberculin by mouth alluded to, Med. 196

Koplik's spots, absence of, in rubella, Epid. 111

—— ——, diagnostic importance in measles, Epid. 110, 111, 130

Kroemer, on separation of divided bones in pubiotomy, alluded to, Obst. 28

Labour (difficult), case of (H. R. Andrews and R. D. Maxwell), Obst. 138

—— ——, case of, treatment by abdominal panhysterectomy, Obst. 140

—— ——, value of crotchet in, Obst. 149

—— (premature), after enucleation, during seventh lunar month of pregnancy, of uterine fibromyoma (H. Williamson), Obst. 73

Labyrinth, anatomy of, Otol. 38

——, extirpation of (S. R. Scott), Otol. 5

—— ——, in labyrinthitis, Otol. 65

——, histological specimens of (lantern slides), demonstrating streptococci in situ from case of otitic meningitis (S. R. Scott), Otol. 157

——, human, histological preparations of (S. R. Scott), Otol. 94

——, necrosis of, specimens from case (P. Macleod Yearsley), Otol. 131

—— of reptiles, birds and mammals, stereoscopic photographs illustrating (A. A. Gray), Otol. 164

——, operations on, risk of, Otol. 156

—— ——, specimens of temporal bone illustrating (C. E. West), Otol. 94

——, pathological conditions found in, specimens and photographs of (A. Gray), Otol. 93

—— (right), suppuration in; operation; recovery (M. Yearsley), Otol. 92

Labyrinthine capsule, Otol. 51

Labyrinthitis, association of middle ear disease with, Otol. 52

Labyrinthitis, histological sections from cases, Otol. 51
——, infection in, path of, Otol. 52
—— ——, spread of, Otol. 51
——, infective organisms in, Otol. 49
——, morbid appearances and course of, Otol. 50
——, sequestration in, Otol. 53, 67
——, symptoms of, Otol. 54
——, tests for presence of, Otol. 70
——, traumatism in causation of, Otol. 50
——, treatment by operation (C. E. West and S. R. Scott), Otol. 37-73
—— —— ——, caution necessary, Otol. 78
—— —— ——, indications, Otol. 71, 77, 78, 81
—— —— ——, methods and records of cases, Otol. 56-70
—— —— ——, results, Otol. 72
——, tuberculous disease in, Otol. 53, 59, 65, 77
—— (acute diffuse), Otol. 53
—— (chronic), Otol. 53
—— —— infective, Otol. 52
Lack, H. Lambert.—Discussion on cases of multiple telangiectases, Laryng. 46
——, discussion on neoplasm of right tonsil, Laryng. 141
—— —— on proptosis due to distension of frontal sinus, Laryng. 50
—— and Green, A. Stanley.—Case of subglottic laryngeal tumour, Laryng. 90. [For discussion see under *Green*, A. Stanley]
Lactation, transmission of bromide eruption from mother to infant through, Derm. 73
Lactic acid in urine of epileptics, Path. 132
—— —— in urine and blood in eclampsia, Path. 131
Laennec, his description of acute suffocative catarrh quoted, Med. 151
Lake, Richard.—Discussion on case of vertigo, Otol. 29
——, discussion on operative surgery of labyrinthitis, Otol. 78
—— —— on specimens and photographs of pathological conditions found in the labyrinth, Otol. 93
——, Menière's disease in a girl aged 14, Otol. 24; discussion (p. 26): Mr. Cheatle, Mr. West, Dr. Dan McKenzie, Mr. C. H. Fagge, Mr. Lake (reply)
——, notes on the effect of treatment in a case of sudden deafness occurring whilst the patient was under treatment for tertiary syphilis, Otol. 7; discussion (p. 8): Mr. Cresswell Baber, Dr. Dundas Grant, Dr. Milligan, Dr. A. Bronner, Dr. Albert Gray, Mr. E. B. Waggett, Mr. A. Cheatle, Dr. Urban Pritchard, Mr. A. L. Whitehead, Dr. Dan McKenzie, Mr. Sydney Scott, Dr. McBride, Mr. Lake (reply)
—— and Gibson, George.—Notes of a case of ablation of both vestibules for the relief of vertigo, Otol. 150; discussion (p. 154): Dr. W. Milligan, Dr. Albert Gray, Mr. Sydney Scott, Mr. Hunter Tod, Dr. Syme, Mr. A. L. Whitehead, Dr. Kerr Love, Dr. McBride, Dr. Fraser (reply)
Land crab, crusade against, instituted by Isthmian Canal Commission, Epid. 313
Landergren, analyses of excretion after restricted diet, quoted, Path. 140
Langmead, F. S., and Caley, H. A.—Statistics of complications of pneumonia from St. Mary's Hospital (1896-1905), Med. 69
—— and Forbes, J. Graham.—Fatal lymphocythæmia in early life, Clin. 129-177; discussion (p. 177): Sir T. Barlow, Dr. Parkes Weber, Dr. Poynton, Dr. Forbes (reply), Dr. Langmead (reply)
Lapwing, advanced tuberculosis found in wild specimen of, Path. 22
Larvæ, passage through intestines, Path. 147
Laryngitis, tuberculous, treated by thyrotomy; case exhibited one year and nine months after operation (H. Barwell), Laryng. 7
Larynx, "bridle" formation in (H. Tilley), Laryng. 16
——, cancer of, treated by laryngectomy (C. J. Symonds), Clin. 184

Larynx, curious manifestations of, in case of hysteria (Sir F. Semon), Laryng. 133
——, disease of (P. Watson Williams), Laryng. 119
—— ——, cases exhibited for diagnosis (F. Spicer), Laryng. 11; (R. H. Scanes Spicer), Laryng. 12
——, excision of, complete, case showing improved method of speaking three years and nine months after (C. J. Symonds), Clin. 184
—— ——, with complete transverse resection of pharynx for malignant growth of posterior pharyngeal wall (W. S. Handley), Clin. 66
——, gumma of (L. Jones), Laryng. 19
——, impaction of part of breastbone of chicken in, for nearly forty-eight hours (F. A. Rose), Laryng. 5
——, leprosy of, specimen (A. Evans), Laryng. 16
——, malignant disease of (D. McKenzie), Laryng. 131
——, neoplasm of, in man aged 75 (R. Scanes Spicer), Laryng. 81
——, nose and pharynx, lesions in, in case of leprosy (StClair Thomson), Laryng. 74
——, operations within, new instrument to facilitate (C. Horsford), Laryng. 24
——, paralysis of, case (H. Clayton Fox), Laryng. 107
——, polypoid growth of (H. B. Robinson), Laryng. 88
——, subglottic tumour of (A. S. Green and H. L. Lack), Laryng. 90
——, tuberculosis of (W. Hill), Laryng. 64
—— ——, extensive, completely cicatrized after treatment with galvano-cautery and tracheotomy (StC. Thomson), Laryng. 9
—— —— and syphilis in, probable combination (Sir F. Semon), Laryng. 135
——, ulceration of (P. Watson Williams), Laryng. 23
—— and pharynx, extensive adhesions in, of syphilitic origin (H. B. Robinson), Laryng. 67
—— ——, endothelioma involving lower part of (H. B. Robinson), Laryng. 68
—— and tonsil, lupus of (J. Dundas Grant). Laryng. 78
——, see *Forceps*, laryngeal : *Intra-laryngeal* operations
Lassalle, Dr., and Dickson, Dr., account of origin of small-pox epidemic in Trinidad, 1902-4 quoted, Epid. 230
Lateral sinus, disease of, operation ; cure (W. S. Syme), Otol. 123
—— ——, phlebitis of, in case of middle ear suppuration (P. McBride), Otol. 135
—— ——, thrombosis of, leucocytosis in, Otol. 137, 138, 139, 142
—— —— ——, followed by meningitis serosa, recovery (H. Tod), Otol. 30
—— —— —— pneumonia and pericarditis, ligature of jugular vein ; complete recovery (W. Permewan), Otol. 129
LATHAM, ARTHUR.—Caseating pulmonary tuberculosis treated by tuberculin (T.R.) and fresh horse serum, both administered by the mouth, Clin. 100 ; discussion (p. 103) : Dr. Parkes Weber, Dr. Theodore Williams, Dr. Latham (reply)
——, discussions on complications of pneumonia, Med. 35
——, preliminary communication on the administration of tuberculin (T. R.) and other vaccines by the mouth, together with (*a*) normal saline solution, (*b*) fresh horse serum, made in conjunction with H. D. Spitta and A. C. Inman ; from the wards and clinical laboratories of St. George's Hospital and the Brompton Hospital for Consumption, Med. 195-239 ; discussion (p. 239) : Dr. F. W. Forbes-Ross ; adjourned discussion (pp. 241-252) : Dr. E. C. Hort, Dr. Hector Mackenzie, Dr. A. G. Auld, Dr. H. D. Rolleston, Dr. H. J. Johnston-Lavis, Dr. Vere Pearson, Mr. Stuart-Low, Dr. R. H. Hodgson, Dr. Latham (reply)
LAW, W. J.—On the termination of the nerves in the teeth of mammalia, Odont. 45 ; discussion (p. 57) : Mr. Hopewell-Smith, Dr. E. I. Spriggs, Mr. Howard Mummery, Mr. W. J. Law (reply)
LAWRENCE, L. A.—Case of a deaf-mute, aged 11, who is apparently regaining her hearing, Otol. 13 ; discussion (p. 13) : Mr. Cheatle, Dr. Dundas Grant, Mr. Lawrence (reply)
——, discussion on case of stenosis of right auditory meatus, Otol. 14

Lawrence, T. W. P.—Ovarian dermoid with secondary cysts connected with the omentum (Martin Randall); with pathological reports and remarks by T. W. P. Lawrence, Obst. 105, 108

Lazarus-Barlow, W. S.—The relation of endothelioma to other forms of new growth, Path. 167

Lead poisoning, chronic constipation due to, Med. 137

Leaf, Cecil.—Discussion on lymphangioplasty for brawny arm of breast cancer, Clin. 189

Leathem, A. N., and MacLeod, J. M. H.—Case of glossitis in a girl aged 3½, Derm. 51

Leathes, J. B.—Acidosis in pregnancy, Path. 131; discussion (p. 144): Dr. Beddard, Dr. Bainbridge, Dr. Leathes (reply)

Lediard, H. A.—Multiple tumours of the skin of doubtful nature, Clin. 18; discussion (p. 20): Sir Dyce Duckworth, Dr. Stowers, Mr. Godlee

Ledingham, J. C. G. and A., cases of typhoid "carriers" referred to, Epid. 212, 213

Leduc, Professor S., first to establish principles of ionic medication on scientific basis, Electr. 70

Leduc's interrupter, Electr. 116, 117

Lees, D. B.—Discussion on diagnostic value of Röntgen rays in some diseases of chest, Electr. 48

—— —— on rheumatism and chorea in childhood, Therap. 82

—— —— on tone of cardiac muscle, Med. 270, 273

Legs, telangiectases (spreading) of feet and (Sir M. Morris and S. E. Dore), Derm. 89

Leiomyoma of skin (multiple), case (J. M. H. MacLeod), Derm. 50

Leitch, A., and Courtauld, L.—The occurrence of lumina in malignant tumours, Path. 173

Lemaine and Ferrand on the tuberculin, cutaneous, and ophthalmic reactions, quoted, Path. 76

Leontiasis ossea affecting other than bones of head, Path. 93

Leprosy, case of (T. J. P. Hartigan), Derm. 83

—— (?), case of, for diagnosis (Sir M. Morris for Dr. Kay), Derm. 52

—— of larynx; specimen (A. Evans), Laryng. 16

——, treatment, method of, in Rangoon, Derm. 56

—— —— with nastin, Derm. 83

—— with lesions in nose, pharynx, and larynx (StClair Thomson), Laryng. 74

Leptothricial organisms in aural discharges, Otol. 111

Leptothrix, throat organism in chronic aural discharge, Otol. 115

Leslie, Sir Bradford, on protection of India from plague, quoted, Epid. 85

Leslie, R. Murray.—Two cases of congenital heart disease in adults, Clin. 34; discussion (p. 37): Dr. A. E. Garrod, Dr. Parkes Weber, Dr. Box, Dr. Murray Leslie (reply)

Letulle, on the tuberculin ophthalmic reaction, referred to, Path. 76

Leucocytes: amount of phagocytosis occurring when human and avian tubercle bacilli are presented to normal human leucocytes in serum of patients suffering from tuberculosis after saturation of serum with human and avian bacilli, Path. 60-65

—— in aural discharges, Otol. 100

—— in cases of lymphocythæmia, differential count of, Clin. 162

—— —— ——, number of, Clin. 161

—— (normal washed), presentation of melanin to, in immune serum as compared with normal serum; results respecting increase of phagocytosis of melanin, Med. 178

—— of normal human blood, inhibitive action of serum of birds upon, Path. 68

——, part taken by, in infectious diseases (S. G. Shattock and L. S. Dudgeon), Med. 169-194

——, phagocytosis of, from different cases of *Bacillus coli* infection, Med. 185

—— (washed), of immune blood in normal serum, result of presentation of melanin to, Med. 183, 184

Leucocytosis accompanying acute ulcerative colitis, Med. 161, 163

—— in thrombosis of lateral sinus, Otol. 137, 138, 139, 142

Leucodermia, case of; symptoms of Addison's disease on death; cirrhosis of suprarenals found post mortem (N. Dalton), Clin. 209

—— and sclerodermia, case of (H. G. Adamson), Derm. 43

Leuconychia, case (E. G. Graham Little), Derm. 49
Leucopenia and enlarged spleen accompanying pigmentation (W. P. Herringham), Clin. 33
Leukæmia, splenic polycythæmia analogous to, Clin. 41
——, treatment by Röntgen rays, Electr. 120, 121
—— (spleno-medullary), treatment by Röntgen rays (W. Ironside Bruce), Electr. 56
Levaditi, silver nitrate method of staining *Spirochæta pallida* in sections, Obst. 164, 165, 166
—— —— —— —— —— —— —— ——, attempts of Saling, and others to disprove, Obst. 167, 168, 169
Levator ani muscle, Obst. 50, 51, 206
—— —— —— as means of support of pelvic viscera, Obst. 206-214
—— —— —— —— —— —— ——, evidence for, Obst. 212
—— —— ——, question of degeneration, Obst. 209, 210, 211
—— —— ——, see also *Coccygeus*, *Ilio-coccygeus*, *Pubo-coccygeus*
Lewers, Arthur H. N.—Discussion on bilateral hæmorrhagic ovarian cysts, Obst. 98
——, discussion on removal of cancerous uterus and glands by Wertheim's method, Obst. 64
——, sarcomatous ovarian tumour coexisting with carcinoma of the uterus, Obst. 69
—— —— —— (perithelioma) coexisting with carcinoma of the uterus (ovariotomy, panhysterectomy), Obst. 156
——, uterus removed by Wertheim's method for advanced carcinoma of cervix, Obst. 100
—— —— —— —— —— —— —— ——, report on specimen by R. D. Maxwell, Obst. 102; discussion (p. 104): Mr. Douglas Drew, Dr. Maxwell, Dr. Herbert Spencer (reply)
Lewis, T.—Discussion on Mendelism in relation to disease, Epid. 166
Lichen annularis or granuloma annulare? (case of ringed eruption) (E. G. Graham Little), Derm. 47
—— plano-pilaris (T. D. Savill), Derm. 169
—— ——, case (W. S. Fox), Derm. 45
—— planus, case (G. W. Dawson), Derm. 13
—— ——, acute (E. G. Graham Little), Derm. 33
—— ——, annular, affecting penis and right forearm (J. M. H. MacLeod), Derm. 34
—— ——, case with unusual features (E. G. Graham Little), Derm. 14
—— ——, relation to granuloma annulare, Derm. 149, 150
—— planus verrucosus, treatment by violet light (H. Radcliffe Crocker and G. Pernet), Derm. 38
—— spinulosus, case (J. L. Bunch), Derm. 10
—— —— associated with seborrhoic dermatitis (J. M. H. MacLeod), Derm. 51
Liébault, A. A., advances study and practice of hypnotism in France, Therap. 53
——, slow appreciation of his work, Therap, 53, 54
Liefmann and Nieter, observations on typhoid "carriers" quoted, Epid. 212, 213
Ligament, visceral pelvic, Obst. 215, 216, 217
Ligaments, uterine, anatomical description of, Obst. 46
Ligature of jugular vein in case of infection of lateral sinus; recovery (W. Permewan), Otol. 139, 140, 142
Light, reflex action of, Neur. 74, 75
—— —— ——, connection with infantile nystagmus, Neur. 75
Linear scarification, multiple, in treatment of lupus vulgaris, Laryng. 83
Lip and nose, extensive gummatous ulceration of, with gummatous hepatitis (J. H. Sequeira), Derm. 183
——, chronic inflammation and desquamation of lips (J. M. H. MacLeod), Derm. 4
Lipoma or cystoma of neck (W. Jobson Horne), Laryng. 38
Lipomata, multiple symmetrical (A. Carless), Clin. 233
Liquids, conduction of electricity in, Electr. 67
Lister, Lord, O.M., F.R.S., his operation for double dislocation of shoulder alluded to, Surg. 198, 200
Lithium and iodine ions in treatment of hydrops articuli of knee, Electr. 81
Little, E. G. Graham.—Case for diagnosis, Derm. 49; discussion (p. 49): Dr. Adamson
——, case for diagnosis, Derm. 164

LITTLE, E. G. GRAHAM.—Case of acute lichen planus, Derm. 33; discussion (p. 33): Dr. Radcliffe Crocker

—— —— of Bazin's disease (erythema induratum) in a young woman, Derm. 27

—— —— of bromide eruption, Derm. 73; discussion (p. 75): Dr. Savill, Dr. H. Radcliffe Crocker, Dr. Whitfield

—— —— of (?) dermatitis artefacta, Derm. 83; discussion (p. 85): Dr. Galloway, Dr. Radcliffe Crocker, Mr. Pernet, Dr. Whitfield (reply)

—— —— of folliculitis decalvans (pseudo-pelade of Brocq), Derm. 86; discussion (p. 86): Sir Malcolm Morris, Dr. Radcliffe Crocker

—— —— of granuloma annulare, Derm. 33; discussion (p. 34): Dr. Pringle

—— —— of leuconychia, Derm. 49; discussion (p. 50): Sir Malcolm Morris

—— —— of lichen planus with some unusual features, Derm. 14

—— —— of lupus erythematosus, Derm. 46

—— —— of molluscum contagiosum, Derm. 182; discussion (p. 182): Dr. Colcott Fox

—— —— of morphœa in a woman aged 65, Derm. 3

—— —— of pityriasis rosea, Derm. 182; discussion (p. 182): Sir M. Morris, Dr. Whitfield

—— —— of prurigo of Hebra, Derm. 181; discussion (p. 182): Dr. Colcott Fox

—— —— of pseudo-xanthoma elasticum of Balzer, Derm. 86

—— —— of "pseudo-pelade of Brocq," Derm. 165

—— —— of "ringed eruption" ("lichen annularis," "granuloma annulare"?), Derm. 47; discussion (p. 48): Dr. Pringle, Dr. Radcliffe Crocker, Dr. Galloway, Dr. Graham Little (reply)

—— —— of unusually generalized nævus verrucosus zoniformis, Derm. 87

—— —— of varus nodulosus of Brooke, Derm. 85; discussion (p. 85): Dr. Radcliffe Crocker, Dr. Colcott Fox, Dr. Pringle, Dr. Graham Little (reply)

——, discussion on case of ringed eruption on hand, Derm. 65

—— —— on ionic medication, Electr. 79

—— —— on tuberculides in girl aged 11, Derm. 175

——, granuloma annulare (with bibliography), Derm. 95-163; discussion (p. 163): Dr. H. Radcliffe Crocker, Dr. James Galloway, Dr. George Pernet, Dr. T. Colcott Fox, Dr. H. G. Adamson, Dr. Graham Little (reply)

——, sections from a case of urticaria pigmentosa in an adult, Derm. 165

——, two cases of favus of the scalp, Derm. 26

——, well-marked rosacea associated with phlyctenular conjunctivitis and ulceration of the eyes, Derm. 181; discussion (p. 181): Dr. Whitfield

LITTLE, J. FLETCHER.—Case of bulbar paralysis with unusual position of the head, Neur. 70

Liver, enlargement of, in cases of lymphocythæmia, Clin. 159

——, post-mortem conditions in cases of lymphocythæmia, Clin. 166

——, see also *Yellow atrophy*

LLOYD-WILLIAMS, E.—Discussion on movements of mandible, Odont. 96

LOCKWOOD, C. B.—Discussion on cystic tumour of suprarenal body, Surg. 218

LOCKYER, C.—Case of sarcoma of the uterus (with report of Pathology Committee), Obst. 245; discussion (p. 248): Dr. Amand Routh

——, discussion on bilateral tuberculous salpingitis, Obst. 262

—— —— on microscopic sections taken from uterus, Obst. 153

—— —— on primary bilateral carcinoma of ovary, Obst. 244

—— —— on removal of cancerous uterus and glands by Wertheim's method, Obst. 65

—— —— on uterine fibroids showing sarcomatous degeneration, Obst. 152

——, pregnancy in the wall of a tubal pus sac, Obst. 248; discussion (p. 251): Dr. Eden, Dr. Tate, Dr. Amand Routh, Dr. McCann, Dr. Lockyer (reply)

Lodge, Sir Oliver, on depth of penetration of ions, quoted, Electr. 69

Loeb and Githens, experiments on production of arteriosclerosis, quoted, Therap. 34

London, opposition to mesmerism in, during earlier half of nineteenth century, Therap. 50

London Hospital, statistics of complications of pneumonia at (1897-1906) (W. J. Hadley), Med. 57

Longridge, C. Nepean.—Creatinin excretion in lying-in women, Obst. 193 ; discussion (p. 194) : Dr. H. Spencer

——, discussion on physiological action of placenta, Obst. 12

Lordosis (extreme) of two years' standing (H. C. Thomson), Neur. 57

Love, J. Kerr.—Discussion on mastoid operation, Otol. 147

——, discussion on thrombosis of lateral sinus, Otol. 141

—— —— on vestibulotomy, Otol. 156

Lumbar puncture, diagnostic value, Surg. 143, 144

—— ——, possible uses combined with injection of antiseptic fluids in treatment of cerebro-spinal meningitis, Surg. 161

—— —— —— —— in treatment of otitic meningitis (A. E. Barker), Clin. 74 ; Surg. 143-162

—— —— —— —— —— —— ——, report of cases, Clin. 74 ; Surg. 146-159

—— ——, procedure of, Surg. 145

—— ——, value of, in treatment of uræmia, Med. 103

Lumière autochrome plate, exhibition of colour photographs taken on (G. Allpress Simmons), Electr. 34

Lumina, occurrence in malignant tumours (W. S. Lazarus-Barlow, L. Courtauld and A. Leitch), Path. 168, 169, 173

Lungs, abscess of, complicating pneumonia, Med. 29

—— ——, complicating pneumonia, fatality, Med. 8, 29

—— —— —— ——, wrong diagnosis of, Med. 29

—— ——, primary, diagnosis confirmed by Röntgen ray examination, Electr. 43

——, gangrene of, complicating pneumonia, Med. 25, 26

—— —— —— ——, fatality of, Med. 7

——, post-mortem condition in cases of lymphocythæmia, Clin. 166

——, œdema of, see *Œdema* (pulmonary)

——, see also *Catarrh* (acute suffocative)

Lupoid ulcer of floor of vestibule of nose (J. Dundas Grant), Laryng. 110

Lupus, case with unusual features suggesting lupus pernio, Derm. 76

—— following tuberculous adenitis, treatment by Röntgen rays combined with cauterization, Electr. 88

—— of epiglottis, case (W. Jobson Horne), Laryng. 106

—— of larynx and tonsil, case (J. Dundas Grant), Laryng. 78

——, treatment by aniline and zinc ions, Electr. 75, 76

—— —— by Röntgen rays, Electr. 121

—— —— —— ——, epithelioma following, Electr. 87, 121

—— (disseminated) with chronic lymphangitis of face and osteo-arthritis of hands (A. Whitfield), Derm. 93

—— erythematosus, case (E. G. Graham Little), Derm. 46

—— —— affecting hands, ears, and scalp (J. M. H. MacLeod), Derm. 88

—— —— —— hands and feet only (H. G. Adamson), Derm. 2

—— —— followed by pneumonia, Med. 23

—— —— in child (J. H. Stowers), Derm. 170

—— —— of face, with condition of fingers simulating Raynaud's disease (J. H. Sequeira), Derm. 17

—— ——, relation to granuloma annulare, Derm. 149

—— ——, treatment by copper ions, Electr. 80

—— —— —— by ionization in five cases (T. J. P. Hartigan), Derm. 45

—— erythematosus saccharo-mycetiform, case (A. W. Williams), Derm. 94

—— vulgaris, plastering lesion after application of X-rays, Electr. 85, 88

—— ——, treatment by Finsen light, Electr. 85

—— —— —— by galvano-cautery, Electr. 84

—— —— —— by multiple linear scarification, Electr. 83

—— —— —— by Röntgen rays, combined with cauterization (J. G. Tomkinson), Electr. 83

—— —— —— by scraping, Electr. 84

Lupus vulgaris, multiple, following measles (J. M. H. MacLeod), Derm. 6, 15
Luys, revival of fallacies of mesmerists by, Therap. 55
Lymphangioma of face (J. P. Roughton), Clin. 91
—— circumscriptum, case for diagnosis (E. G. Graham Little), Derm. 49
——, see also *Polypoid growth*
Lymphangioplasty for brawny arm of breast cancer (W. S. Handley), Clin. 186
Lymphangitis of face (chronic), with disseminated lupus (A. Whitfield), Derm. 93
Lymphatic glands, enlargement of, in rubella, Epid. 103
—— —— from human subject, inoculation of pigeons with salt suspension of, Path. 30
—— ——, part played by, in causation of lymphocythæmia, Clin. 176
—— ——, post-mortem condition in cases of lymphocythæmia, Clin. 165
—— ——, wide-seated invasion of, in case of sarcoma of rectum, Path. 124
Lymphocytes in aural discharges, Otol. 101
——, small, found in excess over large, in lymphocythæmia, Clin. 176
Lymphocythæmia, diagnosis rests on differential count of leucocytes, Clin. 162
——, etiology of, theories respecting, Clin. 169
—— in early life (J. G. Forbes and F. S. Langmead), Clin. 129-177
—— —— ——, cases, blood-condition, 135, 140, 143, 144, 147, 150, 153, 154, 156, 157, 160, 174, 175
—— —— —— ——, clinical features, Clin. 159
—— —— —— ——, histories, Clin. 134, 136, 138, 139, 142, 144, 146, 149, 152, 154, 155, 157
—— —— —— ——, post-mortem examinations, Clin. 135, 137, 138, 140, 143, 145, 147, 150, 153, 155, 156, 157
—— —— —— fatal invariably, Clin. 129
LYSTER, C. R. C.—Discussion on diagnostic value of Röntgen rays in some diseases of the chest, Electr. 50
——, discussion on X-ray diagnosis of renal and ureteral calculi, Electr. 34

MABEN, THOMAS.—Discussion on arteriosclerosis, Therap. 37
——, discussion on tropical diseases and remedies, Therap. 28
MACAN, Sir ARTHUR.—Discussion on case of tubercle of cervix uteri, Obst. 67
——, discussion on removal of cancerous uterus and glands by Wertheim's method Obst. 65
—— —— on the supports of pelvic viscera, Obst. 60
MCBRIDE, PETER.—Discussion on case after operation for angina Ludovici, Laryng. 130
——, discussion on cases after the radical maxillary antrum operation, exemplifying simplification of the after-treatment, Laryng. 59
—— —— on case for diagnosis, Otol. 27
—— —— on case of complete occlusion of posterior nares, Otol. 133
—— —— on cases of epithelioma of ear, Otol. 16
—— —— on case of functional aphonia, Laryng. 80
—— —— —— of intrinsic laryngeal neoplasm in a man aged 75, Laryng. 81
—— —— —— of lupus of epiglottis, Laryng. 106
—— —— —— of partial occlusion of both anterior nares by a congenital cutaneous web, Laryng. 67
—— —— —— of primary epithelioma of tympanum, Otol. 36
—— —— —— of venous angeioma of the soft palate, Laryng. 73
—— —— —— of vertigo, Otol. 30
—— —— on clinical pathology of aural discharges, Otol. 122
—— —— on deafness resulting from epidemic cerebro-spinal meningitis, Otol. 86
—— —— on fracture through temporal bone, Otol. 11
—— —— on histological preparations of human cochlea, Otol. 19
—— —— on injury to pharyngeal portion of Eustachian tube from operative procedures, Otol. 127
—— —— on large post-nasal polypus simulating naso pharyngeal fibroma, Laryng. 103

McBride, Peter.—Discussion on malignant growth of septum and naso-pharynx removed by external osteoplastic operation, Laryng. 118
—— —— on operative surgery of labyrinthitis, Otol. 79, 80
—— —— on pulsating growth in left external auditory meatus, Otol. 33
—— —— on sudden deafness in case of syphilis, Otol. 10
—— —— on unilateral hysterical nerve deafness, Otol. 128, 129
—— —— on vestibulotomy, Otol. 157
——, middle ear suppuration; phlebitis of lateral sinus; cerebellar abscess, Otol. 135-142. [For discussion see *Permewan*, W.—case of infection of lateral sinus]
——, presidential address at opening of Otological Section, Otol. 1
McCann, F. J.—Case of bilateral tuberculous salpingitis, with remarks on the reatment of genital tuberculosis in the female, Obst. 259; discussion (p. 262): Mrs. Boyd, Dr. Lockyer, Dr. Macnaughton Jones, Dr. Herbert Spencer, Dr. McCann (reply)
——, discussion on pregnancy in wall of tubal pus sac, Obst. 252
——, grape-like sarcoma of the cervix uteri (with references), Obst. 253
——, paravaginal fibro-myoma weighing 2½ lb., Obst. 321; discussion (p. 322): Dr. Herbert Spencer, Dr. McCann (reply)
——, pregnancy complicated by a fibroid showing "red degeneration," Obst. 315; discussion (p. 319): Dr. Amand Routh, Dr. Blacker, Dr. Herbert Spencer, Dr. McCann
McCrae, T., cases of leukæmia mentioned, Clin. 132
Macewen, Sir W., views on regeneration of bone quoted, Surg. 235-236
McHattie, Thomas J. T.—Case of placenta prævia with delivery of complete gestation sac at the twenty-eighth week, Obst. 314; discussion (p. 315): Dr. Herbert Spencer, Dr. A. Routh, Dr. McHattie (reply)
McKay, R.—Supernumerary teeth, Odont. 129
Mackenrodt on the supports of the pelvic viscera, quoted, Obst. 56
McKenzie, Dan.—Case in which an endothelioma had been removed from the hard palate (with specimen), Laryng. 62; pathological report (W. Wingrave), p. 62
——, case of malignant disease of the larynx, Laryng. 131; discussion (p. 131): Sir F. Semon, Dr. McKenzie (reply)
—— —— of mucocele of the anterior ethmoidal cell of the left side which has been opened from the nose, Laryng. 101; discussion (p. 102): Dr. Dundas Grant, Mr. Kelson, Dr. McKenzie (reply)
—— —— of suppurating hæmatoma of the septum nasi, with a fistulous opening in the gingivo-labial recess, Laryng. 92; discussion (p. 93): Dr. J. B. Ball, Dr. McKenzie (reply)
——, discussion on bony outgrowths from maxilla and mandible, Laryng. 95
—— —— on case of chronic eczema of both auricles and meatuses, Otol. 132
—— —— —— of Menière's disease, Otol. 26
—— —— —— of unilateral paralysis of tongue, Laryng. 54
—— —— on clinical pathology of aural discharges, Otol. 121
—— —— on cyst on floor of nose, Laryng. 97
—— —— on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 104
—— —— on localized pulsating growth of middle turbinate body, Laryng. 1
—— —— on neoplasm of right tonsil, Laryng. 142
—— —— on pulsating angeioma of nose, Laryng. 142
—— —— on subglottic laryngeal tumour, Laryng. 91
—— —— on sudden deafness in case of syphilis, Otol. 9
—— —— on unilateral hysterical nerve deafness, Otol. 128
——, fistulous opening in the middle line of the neck, just above the hyoid bone, in a woman aged 39, Laryng. 3; discussion (p. 3): Dr. Watson Williams, Mr. De Santi, Dr. Scanes Spicer, Dr. Dundas Grant, Dr. StClair Thomson, Mr. Clayton Fox, Dr. Pegler, Dr. Ball, Dr. McKenzie (reply)
——, paresis of the right vocal cord in a man aged 40, Laryng. 2; discussion (p. 2): Dr. Furniss Potter, Mr. Clayton Fox, Dr. J. B. Ball, Mr. Nourse, Mr. Rose, Mr. Horsford, Dr. StClair Thomson, Dr. McKenzie (reply)

McKenzie, Dan, and Grant, J. Dundas.—Case of median cervical fistula, shown after operation (with microscopic specimen), Laryng. 63; pathological report (W. Wingrave), p. 63; Dr. McKenzie (reply)

Mackenzie, Hector W. G.—Discussion on vaccine treatment by the mouth, Med. 243

——, pneumonia and its complications; an address introductory to a discussion on the subject, Med. 2

——, reply to discussion on complications of pneumonia, Med. 48

Mackenzie, James.—The action of digitalis on the human heart, Therap. 29; discussion (p. 31): Dr. T. E. Burton-Brown, Professor Cushny, Dr. H. C. Cameron, Dr. Mackenzie (reply)

——, discussion on case of angina pectoris with aortitis, Clin. 117

—— —— on tone of cardiac muscle, Med. 270

Mackenzie, Hon. Mr., on value of plague inoculation, Epid. 79

MacLeod, J. M. H.—Case of alopecia areata following small-spored ringworm of the scalp, Derm. 89

——, case of annular lichen planus affecting the penis and right forearm, Derm. 34; discussion (p. 35): Dr. Pringle

—— —— of chronic inflammation and desquamation of lips, Derm. 4

—— —— of "dermatitis papillaris capillitii" (Kaposi), Derm. 166

—— —— of erythema induratum (Bazin) in a woman aged 21, in which a positive ophthalmo-tuberculin reaction had been obtained, Derm. 31; discussion (p. 31): Dr. T. C. Fox

—— —— of grouped comedones in an infant, Derm. 14

—— —— of lichen spinulosus associated with seborrhoic dermatitis, Derm. 51; discussion (p. 51): Dr. Radcliffe Crocker, Dr. Pringle, Dr. Adamson

—— —— of lupus erythematosus affecting the hands, ears and scalp, Derm. 88

—— —— of multiple leiomyoma of the skin, Derm. 50; discussion (p. 50): Dr. Radcliffe Crocker

—— —— of multiple lupus vulgaris following measles, Derm. 6, 15

—— discussion on case for diagnosis, Derm. 38

—— —— —— for diagnosis (granuloma of mouth with penile ulcer), Derm. 58

—— —— —— of chronic X-ray dermatitis of hands, Derm. 80

—— and Collins, E. Treacher.—Two cases of advanced "keratosis follicularis, associated with baldness," Derm. 27

—— and Leathem, A. N.—Case of glossitis in a girl aged 3½, Derm. 51

Macnaughton Jones, H.—Acquired atresia of the introitus and vagina in the adult, Obst. 325

——, carcinoma invading a myoma (with report of Pathology Committee), Obst. 155, 156

——, discussion on bilateral tuberculous salpingitis, Obst. 263

—— —— on case of uterine fibro-myoma, Obst. 78

—— —— on cerebral hæmorrhage in eclampsia of pregnancy, Obst. 92

—— —— on hydrosalpinx and ovarian cyst simulating malignant disease, Obst. 242

—— —— on myomectomy during pregnancy, Obst. 136

—— —— on ovarian dermoid with secondary cysts connected with omentum, Obst. 118

—— —— on physiological action of placenta, Obst. 12

—— —— on pubiotomy, Obst. 29

—— —— on sarcoma of uterus, Obst. 236

—— —— on supports of the pelvic viscera, Obst. 221

—— —— on uterine fibroids showing sarcomatous degeneration, Obst. 152

——, hæmatosalpinx complicating myoma, Obst. 122; discussion (p. 123): Mr. Alban Doran, Dr. Herbert Spencer, Dr. Macnaughton Jones (reply)

——, an improved demonstration pelvis with attached fœtal skull for demonstrating to students the mechanism of labour, Obst. 41

——, primary unilateral tuberculosis of the Fallopian tube, Obst. 177; discussion (p. 187): Mr. Spanton, Dr. Macnaughton-Jones (reply)

Macphail, Alexander.—Discussion on the supports of the pelvic viscera, Obst. 221

Macpherson, Lieut.-Col., R.A.M.C.—Discussion on medical inspection in schools, Epid. 60

——, discussion on plague, Epid. 94

—— —— on preventive medicine at Panama, Epid. 316

McQuade, H., specimens exhibited from his case of intestinal myiasis, Path. 148
Macroglossia neurofibromatosa, case of (W. G. Spencer and S. G. Shattock), Path. 8
Macula, changes at, in cerebral degeneration in four members of family (M. S. Mayou), Neur. 98
Magnesia, value in dyspepsia, Therap. 10
Magnesium ions in treatment of warts, Electr. 75
Malaria, Therap. 22
——, cause of unhealthiness and mortality at Isthmus of Panama, Epid. 304
——, disappearance from England, how accounted for, Therap. 24
——, human evolution against, Odont. 113
——, mortality of British Army from, in West Indies (1780-96), Epid. 304, 305
—— parasite, extra-corporeal life in mosquito, Therap. 23
—— —— of, potency of quinine in attacking, Therap. 23
—— —— —— —— —— ——, when limited, Therap. 23
——, prevalence in Mauritius, Epid. 316
——, preventive measures against, instituted by Isthmian Canal Commission, Epid. 312
——, ravages of, in Sudan, Epid. 314
——, suppression of, at Klang and Port Swettenham (Federated Malay States), Epid. 315
—— —— by means of mosquito destruction, Epid. 316, 317
——, treatment by quinine in combination with other drugs, Therap. 23, 24
Malay States (Federated), suppression of malaria at stations in, Epid. 315; see also *Klang* and *Port Swettenham*
MALCOLM, J. D.—Carcinoma of both ovaries and of the sigmoid flexure, Obst. 236 (with report of Pathology Committee, p. 240)
——, cases of cholecystectomy, Surg. 93-105; discussion (p. 105): Mr. Moynihan
——, discussion on case of chorionepithelioma of uterus, Obst. 17
——, large hydrosalpinx and small ovarian cyst simulating malignant disease in a woman, aged 58, Obst. 241; discussion (p. 242): Dr. Herbert Spencer, Dr. Macnaughton Jones, Mr. Malcolm (reply)
——, twisted inflamed Fallopian tube, Obst. 99
Male sex, hæmophilia practically limited to, Epid. 153
Malgaigne, cases of swelling of callus formed after fractures, quoted, Path. 109
Malignancy, high degree of, indicated by presence of pigmentation in vulval and vaginal tumours, Obst. 127
Malignant disease, growth of, in man and lower animals, with special reference to vascular system (Prof. E. Goldmann), Surg. 1
—— ——, large hydrosalpinx with small cyst of ovary simulating (J. D. Malcolm), Obst. 241
—— —— of thyroid, operation results unsatisfactory, Surg. 36
—— ——, treatment by Röntgen rays, Electr. 120
—— ——, ulceration and destruction of epiglottis (W. Jobson Horne) Laryng. 33
—— growth behind cricoid cartilage (J. W. Bond), Laryng. 111
—— —— of septum and naso-pharynx removed by external osteoplastic operation (P. Watson Williams), Laryng. 115
—— —— (ulceration) of inferior turbinated body (E. Cresswell Baber), Laryng. 41
—— growths, blood-vessels in, purpose of their multiplication, Surg. 10
—— ——, circulation in, Surg. 5
—— ——, dissemination along vascular system, Surg. 1
—— ——, lumina in (L. Courtauld and A. Leitch), Path. 168, 173
—— ——, treatment, individualization advocated in preference to stereotyped surgical interference, Surg. 12
Malingering, see *Ulcers* (factitious)
Malnutrition, factor in increase of insanity, Odont. 119, 123
Malpighian or basal squamous cell carcinomata, Path. 168
Mammalia, labyrinth of, stereoscopic photographs illustrating (W. G. Spencer), Surg. 187
——, teeth of, termination of nerves in (W. J. Law), Odont. 45

Manchot, suggestion as to cause of arteriosclerosis, quoted, Therap. 35

MANDERS, HORACE.—Discussion on presidential survey of electro therapeutics, Electr. 14

Mandible, development of, Odont. 18

——, growth of, influenced by growth of upper jaw, Odont. 23

——, movements of, Odont. 79

——, necrosis of, extensive, case of (W. W. James), Odont. 29

——, relation of teeth-bearing to other parts of, Odont. 23

—— and maxilla, bony outgrowths from (W. H. Kelson), Laryng. 91

Manganese, black oxide of, in treatment of irritable dyspepsia, Therap. 19

MANN, H. C. C., **FAWCETT, JOHN**, and CAMERON, H. C.—Statistics of complications of pneumonia from Guy's Hospital (1902-6), Med. 69

Manson, Sir Patrick, study and recognition of the host of filaria the basis of his mosquito-malaria theory, Therap. 25

Marriages, consanguineous, relation of Mendelism to effects of, Epid. 162

Marshall, H. T., on the causation of endocarditis by tubercle bacilli, quoted, Path. 121

MARTIN, C. J., F.R.S.—Discussion on etiology and epidemiology of typhoid fever, Epid. 220

—— discussion on plague, Epid. 96

MARTIN, J. MIDDLETON.—Discussion on medical inspection in schools (contribution by letter), Epid. 62

Massage in treatment of dyspepsia of central nervous origin, Therap. 8

—— —— —— of chorea mollis, Therap. 77

Masson, Dr., discovery of small-pox epidemic in Trinidad (1902) due to, Epid. 234

MASTERS, A.—Discussion on hairball in the stomach, Clin. 214

Mastication, efficient, importance of foodstuffs requiring, in prevention of dental caries, Odont. 37

—— —— —— insistence on, in children in prevention of dental caries, Odont. 37

Mastoid disease associated with labyrinthitis, Otol. 57, 63, 67, 70, 78, 82

——, empyema of, Bezold's, discharging into pharynx (W. S. Syme), Otol. 125

—— operation, use of fresh horse serum as dressing for, Med. 248

—— —— with preservation of tympanic membrane and ossicles, patients shown after (A. Logan Turner), Otol. 144

—— ——, radical, after-treatment by partial grafting with aid of local anæsthesia (J. Stoddart Barr), Otol. 145, 148

—— —— ——, unusual sequel to (W. G. Porter), Otol. 144

—— process, pus reaching from antrum to neck without passing through (A. H. Cheatle), Otol. 24

—— ——, swelling of, case for diagnosis (R. S. Cocke), Otol. 27

—— region, secondary infection of skin over, in case of primary infection of tympanic cavity on left side (H. Tod), Otol. 15

—— suppuration, acute, operation for, followed by keloid (H. Tilley), Otol. 20

Maton, W. G., first accurate description of rubella by, Epid. 117

Matrix of aural discharges, Otol. 113

Mauritius, prevalence of malaria in, Epid. 316

Maxilla, see also *Jaw*

—— and mandible, bony outgrowths from (W. H. Kelson), Laryng. 91

Maxillæ, upper, bilateral sarcoma of (A. Hutchison), Laryng. 21

—— —— —— swelling of, report of Morbid Growths Committee on, Laryng. [illegible]

Maxillary sinus, see *Antrum* (maxillary)

MAXWELL, J. PRESTON.—Case of "womb stone" (exhibited by J. Abernethy Willett), Obst. 228

MAXWELL, R. D.—Discussion on case of uterine fibromyoma, Obst. 78

—— discussion on intra-uterine death of syphilitic fœtus in six consecutive pregnancies, Obst. 171

——, report on specimen of uterus removed by Wertheim's method for advanced carcinoma of cervix, Obst. 102, 104

Maxwell, R. D., and Andrews, H. R.—Case of difficult labour, Obst. 138; discussion (p. 148): Dr. Champneys, Dr. W. S. A. Griffith, Dr. Herman, Dr. Eden, Dr. Herbert Spencer, Dr. Andrews (reply), Dr. Maxwell (reply)
May, W. J.—Discussion on treatment of pyorrhœa, Odont. 66
Maygrier and Chavane, their case of cerebral hæmorrhage in eclampsia of pregnancy described, Obst. 86
Mayou, M. S.—Cerebral degeneration with changes at the macula in four members of a family, Neur. 98; discussion (p. 99): Dr. Guthrie, Dr. G. Holmes (reply)
——, discussion on case of multiple hereditary developmental angiomata, Clin. 66
—— and Guthrie, Leonard G.—Right hemiplegia and atrophy of left optic nerve, Clin. 180
Measles, age-incidence of, differs from that of rubella, Epid. 109
——, changes and age- and sex-incidence before and during school life, Epid. 58
——, diagnosis of, importance of Koplik's spots in, Epid. 110, 111, 130
—— followed by multiple lupus vulgaris (J. M. H. MacLeod), Derm. 6
——, seasonal prevalence, differs from that of rubella, Epid. 109
Meat-juice, raw, in treatment of tabes mesenterica, Med. 246
Meat poisoning by *B. enteritidis*, Gärtner, comparable to poisoning from eating shellfish, Path. 82
Meatus (auditory), chronic eczema of both auricles and meatuses with unusual degree of hyperplasia (E. Furniss Potter), Otol. 132
—— —— (external), deep ulceration of, with purulent otitis media in case of secondary syphilis (A. Bronner), Otol. 88
—— —— ——, branchial sinus leading into; sinus excised (C. H. Fagge), Otol. 90
—— —— (external), left, pulsating growth in (J. B. Ball), Otol. 32
—— ——, right, stenosis of (W. H. Kelson), Otol. 14
Meckel's cartilage, Odont. 18
Mediastinum, disease of, œdema of hands and feet with mediastinal affection (F. Parkes Weber), Clin. 192
——, tumour of, hydatid cyst? (H. French), Clin. 93
Medical history card supplied to school children, Epid. 61, 62
Medical inspection in schools, Gloucestershire scheme of (M. Coplans), Epid. 45
—— —— —— —— —— ——, mode of, Epid. 49
Medical officers of health, superior status accorded to, in United States, Epid. 314
Medicine (preventive), progress of, at Panama (Sir F. Treves), Epid. 303-313
Medulla, histology of, in section from case of diffuse osteoma, Path. 100
Meek, W. O.—Tuberculous endocarditis, Path. 116
Megalerythema infectiosum, Epid. 112
Melæna at onset of acute ulcerative colitis, Med. 161, 163
Melanin, de-opsonization of immune serum by saturation with, Med. 177, 179, 181
——, opsonization of, Med. 182
——, phagocytosis carried out by means of (S. G. Shattock and L. S. Dudgeon), Med. 169-194
—— ——, amounts of, on presentation of melanin to washed cells of patient and to washed normal cells in immune serum, Med. 184-191
—— ——, increase by adding melanin to blood from subject of infective disease, as compared with normal blood, Med. 173, 177
—— ——, increase compared by presentation of melanin to normal washed leucocytes in immune serum as compared with normal serum, Med. 178
—— ——, question of increase if melanin is presented to washed leucocytes of immune blood in normal serum, Med. 183, 184
——, reduction of phagocytosis of normal cells by saturation of immune serum with melanin, against bacterium, or suspension of melanin, or against both, Med. 179, 181
——, suspension of, heating of immune serum causing reduction of phagocytosis towards, Med. 182
—— ——, preparation, Med. 171

Melanotic tumour of vulva (E. Holland). Obst. 124
—— —— ——, irregularity of pigmentation in, cases. Obst. 126
—— —— ——, rarity of occurrence, Obst. 126
Men, spitting habits among, factor in spread of pneumonia, Med. 49
Mendelism, existence of characters in alternative pairs essential feature of, Epid. 136, 139
——, how far diseases are subject to laws of, Epid. 160
—— illustrated by brachydactyly, Epid. 141
—— —— by diversity of characters, Epid. 139
—— —— by eye colour, Epid. 141
—— —— by human examples, Epid. 141
—— —— by pedigree of night-blindness, Epid. 143, 163, 164
—— in relation to disease (R. C. Punnett), Epid. 135-161
—— —— to state medicine, Epid. 165
——, objections to, stated, Epid. 162-164
——, principles of, Epid. 136
——, relation to effects of consanguineous marriages, Epid. 162
Ménière's disease, see *Vertigo* (aural)
Meningitis complicating pneumonia, Med. 29
——, diagnosis of, by aid of cerebrospinal fluid, Otol. 140, 141
—— (cerebrospinal, epidemic), deafness resulting from; two cases (H. H. B. Cunningham). Otol. 83
—— ——, diagnostic value of lumbar puncture in, Surg. 144
—— ——, treatment by lumbar puncture combined with injection of antiseptic fluids, Surg. 161
—— (otitic), micro-organisms found in, Surg. 145, 151, 156, 158
—— ——, treatment by lumbar puncture (A. E. Barker), Clin. 74, Surg. 143-162
—— —— —— —— —— ——, recovery without injury to nervous centres, Surg. 160
—— —— —— —— —— ——, report of cases, Surg. 146-159
—— ——, with histological specimens (lantern slides) of labyrinth demonstrating streptococci in situ (S. R. Scott), Otol. 157
—— (pneumococcic) complicating pneumonia, Med. 11, 22
—— (posterior basic), treatment of, Surg. 164
—— serosa, following lateral sinus thrombosis (H. Tod), Otol. 30
Meningococcus in aural discharges, Otol. 108
——, see also *Serum* (antimeningococcic)
Menorrhagia, treatment by ionic medication, Electr. 134, 135
Menstruation, influence of pituitary gland on, Obst. 311
—— not a causal factor of fertility, Obst. 294
——, periodicity of, Obst. 309
——, pro-œstrum stage of "rut" of animals homologous with, Obst. 293
——, relation of blood-calcium metabolism to, Obst. 291, 295, 297, 300
—— —— —— ——, experiments and observations on rabbits respecting, Obst. 301, 305
Mental emotion as excitant of epilepsy, Med. 92
MERCIER, CHARLES.—Discussion on mechanism of nystagmus, Neur. 81
Mercury, administration in pregnancy, value of, in preventing syphilis in future offspring, Obst. 163, 170, 171
Mesenteric glands, calcified, causing confusion in X-ray diagnosis of calculi, Electr. 27
Mesmerism encouraged in India, Therap. 50, 51
——, fallacies of, revived by school of the Salpêtrière, Therap. 55
——, operations under, Therap. 50, 51
——, opposition to, in London, Therap. 50
—— —— in Scotland, Therap. 51
——, term discarded for that of "hypnotism" by Braid, Therap. 52
——, see also *Hypnotism*
Mesoblastic cells in aural disharges, Otol. 100

Mesoblastic cells, instability of types of growth originating from, Path. 170
Mesothelial cells in aural discharges, Otol. 101
Metal, conduction of electricity in, Electr. 67
Metastasis (vaginal and visceral), extensive, in case of chorionepithelioma of uterus (T. W. Eden), Obst. 13
—— of tumours, new views respecting, Surg. 3, 4
Meter for measuring Röntgen rays, Electr. 93-96
Methæmoglobinæmia of twelve years standing (W. E. Wynter), Clin. 48
——, see also *Anæmia*, cyanotic
Metronome, clockwork contrivance for control of interrupted currents, Electr. 115
Metz, incidence of typhoid fever at, Epid. 206-209
Meyer-Wirz, his case of cerebral hæmorrhage in eclampsia of pregnancy described, Otol. 86
Mice, possible agency of, in spreading plague, Epid. 93
Michels, E., and Weber, F. Parkes.—Ischæmic disturbance of the right upper extremity, Neur. 100
Micrococcus cæruleus producing tints in aural discharges, Otol. 97
—— *catarrhalis*, presence in cases of otitic meningitis, Surg. 145, 151, 165
—— *fæcalis*, presence in cases of otitic meningitis, Surg. 145
—— *tetragonus* in chronic aural discharges of tuberculous nature, Otol. 116
Mid-brain centre, nystagmus dependent on, Neur. 78
Middlesex Hospital, statistics of complications of pneumonia at (W. Pasteur), Med. 54
Migraine, see *Hemicrania*
Milk as factor in causation of pyorrhœa alveolaris, Odont. 106
——, homogenized, advisable for dyspeptics, Therap. 16
——, transmission of typhoid fever by, through agency of "carrier," Epid. 180
Miller, Reginald.—Pathological report on Mr. H. S. Sington's case of tumour of fœtal head, Obst. 160
Milligan, William.—Demonstration of specimens, Otol. 18
——, discussion on cases of epithelioma of ear, Otol. 16
—— —— on case of extirpation of labyrinth, Otol. 6
—— —— —— of mucocele of frontal sinus, Laryng. 72
—— —— —— of venous angeioma of the soft palate, Laryng. 73
—— —— —— of unilateral nerve deafness, Otol. 130
—— —— on clinical pathology of aural discharges, Otol. 120
—— —— on deafness resulting from epidemic cerebrospinal meningitis, Otol. 87
—— —— on fracture through temporal bone, Otol. 12
—— —— on injury to pharyngeal portion of Eustachian tube from operative procedures, Otol. 126
—— —— on keloid following mastoid operation, Otol. 21
—— —— on lateral sinus thrombosis, Otol. 140
—— —— on mastoid operation, Otol. 147
—— —— on operative surgery of labyrinthitis, Otol. 75
—— —— on primary epithelioma (?) of tympanum, Otol. 35
—— —— on pulsating growth in left external auditory meatus, Otol. 33
—— —— on sudden deafness in case of syphilis, Otol. 8
—— —— on unilateral hysterical nerve deafness, Otol. 129
—— —— on vestibulotomy, Otol. 154
——, epithelioma of middle ear (traumatic?), Otol. 91; discussion (p. 92): Dr. Paterson, Dr. Milligan (reply)
Mixed infections complicating pneumonia, Med. 15
Molar (first), position of, determination of relation of teeth to one another by, Odont. 143
—— (upper third), see *Wisdom tooth*, upper
—— of horse, effects of chronic suppuration in (J. G. Turner), Odont. 26
Molars (deciduous), removal of, in prevention of oral sepsis, Odont. 38, 39, 70, 72

Molars (temporary), incomplete eruption of (H. W. Trewby), Odont. 99
Mollendorff on association of migraine with epilepsy, Med. 89
Molluscum contagiosum (E. G. Graham Little), Derm. 182
—— —— in adult (J. H. Stowers), Derm. 172
—— —— spread by birds, Derm. 172
Monkey, see *Rhesus monkey*
Monro, on the association of Raynaud's disease with epileptic fits, quoted, Med. 88
Monstrosities, see *Gastroschisis*
Moore, F. Craven.—Pathological report on case of complete gastrectomy, Surg. 88
Moore, Norman.—Myxœdema with optic atrophy, Clin. 194
Morbid Growths Committee (Laryngological Section), report on bilateral swelling of upper maxillæ (A. Hutchison), Laryng. 43
—— —— —— —— —— —— on infiltration of nasal cavities (C. A. Parker), Laryng. 115
—— —— —— —— —— —— on pulsating growth (A. Tweedie), Laryng. 15
Morgenstern, investigations respecting presence of nerve fibres in dentine, quoted, Odont. 48
Morison, Alexander.—Discussion on tone of cardiac muscle, Med. 271
Moro and Doganoff, cutaneous tuberculosis inoculated by v. Pirquet's method, Path. 75
Morphia only available remedy in pain of acute pericarditis, Therap. 71
Morphœa, case of (E. G. Graham Little), Derm. 3
—— —— (J. H. Sequeira), Derm. 90
——, see also *Scleroderma*
Morris, Henry.—Discussion on growth of malignant disease, Surg. 18
Morris, Sir M.—Bullæ (thick-walled) on face; case for diagnosis, Derm. 17
——, case for diagnosis (shown for Dr. Kay), Derm. 52
——, discussion on case of chronic X-ray dermatitis of hands, Derm. 80
—— —— —— of disseminated lupus, Derm. 94
—— —— —— of folliculitis decalvans (pseudo-pelade of Brocq), Derm. 86
—— —— —— of leuconychia, Derm. 50
—— —— —— of macular atrophy of scalp, Derm. 60
—— —— —— of pityriasis rosea, Derm. 182
—— —— on dermatitis herpetiformis of unusual type, Derm. 180
—— and Dore, S. E.—Case of spreading telangiectases of the feet and legs, Derm. 89
—— and Fox, W. S.—Case of von Recklinghausen's disease, Derm. 16
Morton, Reginald.—Discussion on electrolytic method of measuring X-rays, Electr. 99
—— discussion on treatment of leukæmia, exophthalmic goitre, and malignant disease by X-rays, Electr. 60
——, the electrical treatment of atonic conditions of the digestive system, Electr. 101; discussion (p. 106): Mr. Deane Butcher, Dr. Lewis Jones, Dr. Buckley, Dr. David Arthur, Mr. Bokenham, Dr. Pirie, Dr. G. B. Batten, Dr. E. S. Worrall, Dr. Morton (reply)
Morton wave current, Electr. 109, 111
Mosquito, extermination of, in suppression of malaria, Epid. 316, 317
——, extra-corporeal life of parasite of malaria in, Therap. 23
——, trees as breeding-places for, Epid. 312, 316, 317
——, see also *Culex* mosquito
Moths, crossing of, Epid. 163
Motor area of brain, tumour of, operated upon (H. C. Thomson), Neur. 96
Moullin, J. A. Mansell.—Discussion on bilateral hæmorrhagic ovarian cysts, Obst. 98
Mouse, favus in (T. P. Beddoes for P. S. Abraham), Derm. 64
Mouse-tumours, question of nomenclature, Path. 171
Mouth: administration of tuberculin (T.R.) and other vaccines by mouth together with (*a*) normal saline solution, (*b*) fresh horse serum (A. Latham, H. D. Spitta and A. C. Inman), Med. 195-239
——, granuloma of, with penile ulcer (J. H. Sequeira), Derm. 57
——, granulomatous swellings at left angle of mouth and in right inguinal region (J. H. Sequeira), Derm. 92

Mouth, ulceration of, treatment by copper ions, Electr. 75
—— —— round, extensive gummatous (J. H. Sequeira), Derm. 185
Movements (involuntary) in hemiplegia (H. C. Thomson), Neur. 55
Moxon, W., on cardiac arrest in idiopathic epilepsy, quoted, Med. 76, 77
MOYNIHAN, B. G. A.—Case of complete gastrectomy, Surg. 81; pathological report by F. Craven Moore, Surg. 88; discussion (p. 91): Mr. Warrington Haward, Mr. McAdam Eccles
——, discussion on cholecystectomy, Surg. 105
Mucocele of anterior ethmoidal cell of left side opened from nose (D. McKenzie), Laryng. 101
—— of frontal sinus, case of (F. J. Steward), Laryng. 71
Mucous membranes, inflammatory conditions of, complicating pneumonia, Med. 13
—— ——, multiple hereditary developmental angiomata (telangiectases) of skin and mucous membranes (F. Parkes Weber), Clin. 65
MUMMERY, J. HOWARD.—Discussion on difficult extraction, Odont. 138
——, discussion on treatment of children from dental aspect, Odont. 40
—— —— on termination of nerves in teeth of mammalia, Odont. 57, 59
——, presidential address at opening meeting of Odontological Section, Odont. 1
MUMMERY, J. P. LOCKHART.—Case of hemihypertrophy (with bibliography), Clin. 61
——, discussion on case of rupture of brachial plexus at birth, Clin. 219
—— —— on treatment of ulcerative proctititis by zinc cataphoresis, Surg. 185
MUMMERY, STANLEY P.—Discussion on treatment of children from dental aspect, Odont. 75
——, heredity and dental disease, Odont. 108; discussion (p. 119): Mr. F. J. Bennett, Dr. Sim Wallace, Mr. Norman Bennett, Mr. Kenneth Goadby, Mr. H. Baldwin, Mr. Stanley Mummery (reply)
Mumps, changes in age- and sex-incidence before and during school life, Epid. 58
Münzer, on urine excretion in acute yellow atrophy, alluded to, Path. 142
Murchison, Charles, case of leontiasis ossea, quoted, Path. 93
MURPHY, Sir SHIRLEY F.—Discussion on etiology and epidemiology of typhoid fever, Epid. 223
——, discussion on Mendelism in relation to disease, Epid. 166
—— —— on preventive medicine at Panama, Epid. 314
—— —— on rubella, Epid. 124
Murray, G., on convulsive attacks in association with cardiac failure and bradycardia, quoted, Med. 74, 75
MURRAY, WILLIAM.—The therapeutics of indigestion, Therap. 13; discussion (pp. 19-22): Dr. Soper, Dr. Cecil Wall, Dr. J. Gray Duncanson, Dr. Ince, Dr. Herbert French, Dr. Murray (reply)
Muscle, limitation in propagation of excitation by, Odont. 46
——, cardiac, effects of digitalis on conductivity and contractility, Med. 264
—— ——, tone of (A. M. Gossage), Med. 253-270
—— —— ——, clinical accounts of patients from whom tracings were obtained, Med. 268, 269
—— —— ——, definition, Med. 253
—— —— ——, effects of increase, Med. 256, 271
—— —— ——, increase and decrease, effects on conductivity and contractility, Med. 254-258, 271
—— —— ——, means for restoration, Med. 258
—— ——, see also *Myasthenia* (cardiac)
Muscle-fibres of vessels essential seat of arteriosclerosis, Therap. 36
Muscle-reflex action, Neur. 72
—— —— centre, influence of light on, Neur. 75
Muscle-spindles in ocular muscles, Neur. 73
—— —— (E. F. Buzzard), Neur. 83
Muscles (abdominal), permanent paralysis of, resulting in case of acute anterior poliomyelitis (W. Pasteur), Clin. 51
——, acting and antagonistic, Neur. 71
—— behind shoulder, normal position with outstretched arm, Surg. 190, 192

Muscles (ocular), muscle-spindles in, Neur. 73
—— —— —— —— (E. F. Buzzard), Neur. 83
—— ——, neural spindles in, Neur. 73
—— of pelvic diaphragm, Obst. 49, 200
—— (opposing), alternate contraction in hemiplegia, Neur. 73
—— —— —— —— ——, automatic actions of, Neur. 72
—— (pectoral), congenital bilateral absence of, only one case on record, Clin. 73
—— —— —— unilateral absence of (J. G. Forbes), Clin. 71
—— —— —— —— —— ——, etiology, Clin. 73
—— (perineal, superficial), anatomical description of, Obst. 48
——, "periodic time" of, Electr. 104, 110
—— (small) of hand, wasting of, in girl aged 20, and her father aged 46 (T. Thompson), Neur. 63
——, toxic affection of muscles and lower neurones (C. E. Beevor), Neur. 2
Muscular system, congenital defect of (R. Howard), Path. 157
—— —— —— —— ——, association with congenital talipes equino-varus (R. Howard), Path. 157
—— —— —— —— ——, microscopical examination of sections post mortem, Path. 162
Museum of Odontological Section, specimens added to, described (J. F. Colyer), Odont. 27
Museum specimens of coxa valga, Surg. 110, 115
Mutton, well-kept and roasted, necessity of, for easy digestion, Therap. 16
Myasthenia (cardiac), Med. 272
Myatonia congenita, case (C. E. Beevor), Neur. 1
Mycosis fungoides (H. Radcliffe Crocker), Derm. 167
—— ——, effect of X rays on (H. Radcliffe Crocker), Derm. 75
Myelocytes in aural discharges, Otol. 102
Myiasis, intestinal, see *Eristalis*; *Intestine*
Myocarditis, treatment by digitalis unavailing, Therap. 70
Myoma, carcinoma invading, specimen of (H. Macnaughton Jones), Obst. 155
—— —— —— ——, report of Pathology Committee on, Obst. 156
—— of uterus complicated by hæmatosalpinx (H. Macnaughton Jones), Obst. 122
——, see also *Leiomyoma*
Myomectomy in treatment of fibroid tumours complicating pregnancy, Obst. 318
—— during pregnancy, postponement till viability, desirable, Obst. 135, 137
—— (abdominal), during pregnancy (W. C. Swayne), Obst. 129
—— —— —— ——, indications for, Obst. 131
—— ——, result on scar left in uterine tissue after, Obst. 131
—— ——, with supravaginal hysterectomy in case of pregnancy complicated with large retro-peritoneal and subligamentous fibromyoma (J. H. Dauber), Obst. 132, 133
Myopathy, case of (B. Abrahams), Clin. 24
—— —— (J. Taylor), Neur. 7
Myositis ossificans (C. A. R. Nitch), Clin. 107
Myristicin, active poisonous principle of nutmeg, Therap. 40, 41, 43
Myxœdema, association of atrophy of thyroid gland with, importance of discovery, Path. 1
——, case with unusual features (A. M. H. Gray), Clin. 26
—— with optic atrophy (N. Moore), Clin. 194

Nævus, verrucose (W. S. Fox), Derm. 12
—— verrucosus zoniformis, generalized (E. G. Graham Little), Derm. 87
Nails (diseases of), see *Leuconychia*
Nancy, school of, in hypnotic teaching, Therap. 54
—— —— —— —— —— ——, carried on work of Braid, Therap. 54
—— —— —— —— —— ——, orderly character of, Therap. 64
Nares, see *Nostrils*
Nasal breathing, importance of, in prevention of dental caries, Odont. 38

Nasal obstruction, case (H. J. Davis), Laryng. 75
—— —— on left side (H. J. Davis), Laryng. 139
NASH, J. T. C.—Discussion on rubella, Epid. 130
Naso-pharynx, atresia of (C. Potter), Laryng. 15
——, fibroma of, simulated by large post-nasal polypus growing from antrum of Highmore (J. Dundas Grant), Laryng. 102
—— ——, large pedunculated (? fibromyxoma) removed from vault of (H. J. Davis), Laryng. 138
——, tumour of, removed fifteen weeks after operative removal of adenoids (E. F. Potter), Laryng. 9
—— and oro-pharynx, partial and progressive atresia of, in case of complete closure of anterior nares (W. Stuart-Low), Laryng. 56
—— and septum, malignant growth of, removed by external osteoplastic operation (P. Watson Williams), Laryng. 115
Nastin in treatment of leprosy, Derm. 83
Natural selection, application to artificial conditions, Odont. 121
—— ——, influence on human race, Odont. 109
—— ——, removal of influence of, cause of prevalence of dental caries, Odont. 114-117
—— —— —— —— —— —— —— —— of insanity, Odont. 75, 113
Neck, extensive cellulitis of right side of (P. Abercrombie), Laryng. 10
——, lipoma or cystoma of (W. Jobson Horne), Laryng. 38
——, middle line of, fistulous opening in, just above hyoid bone (D. McKenzie), Laryng.
——, muscles of, post-mortem examination in case of congenital defect of muscular system, Path. 159
—— pus reaching from antrum to, without passing through mastoid process (A. H. Cheatle), Otol. 24
——, subcutaneous induration of, origin uncertain (E. Ward), Laryng. 112
——, see also *Vertebræ*, cervical
Necrosis of arytænoid cartilage, case of (H. Barwell), Laryng. 65
—— of labyrinth, specimen from case (P. Macleod Yearsley), Otol. 131
—— of mandible, extensive (W. W. James), Odont. 29
——, quiet, simulating sarcoma, Path. 93
Negro race, liability to pneumonia, Epid. 310, 316
—— ——, susceptibility of, to small-pox, Epid. 246
Neisser, on staining for *Spirochæta pallida*, quoted, Obst. 168
Neoplasm, intrinsic, of left vocal cord (R. H. Scanes Spicer), Laryng. 81, 89
—— of right tonsil (C. A. Parker), Laryng. 140
Neoplasms, symmetrical, on vocal cords (W. Jobson Horne), Laryng. 13
Nephritis complicating pneumonia, Med. 14, 43, 44
——, chronic, fatal complication of pneumonia, Med. 28
Nerve (facial), Otol. 47
—— (great sciatic), anatomy of, Surg. 167
—— —— ——, removal of adhesions from, in treatment of sciatica, Surg. 171-173, 175
—— (optic), atrophy of (N. Moore), Clin. 194
—— ——, left, atrophy of, with right hemiplegia, Clin. 180
Nerve-areas, primary cutaneous carcinoma of chest involving neighbouring (D. Walsh), Derm. 187
Nerve-deafness, hysterical, unilateral, of sudden onset (J. Dundas Grant), Otol. 127
—— ——, unilateral (J. Dundas Grant) Otol. 130
Nerve-fibres, afferent and efferent, convey excitation in both directions, Odont. 46, 47
—— ——, presence in dentine, Odont. 47, 48, 49
—— —— —— ——, methods employed for demonstration of, Odont. 50-56
Nerve-root grafting (P. Sargent), Neur. 8
—— stretching in sciatica, Surg. 171
Nerves, termination of, in teeth of mammalia (W. J. Law), Odont. 45

Nervous instability and emotional temperament paramount factors in causation of chorea, Therap. 72
Nervous system (central), depressant action of nutmeg on, Therap. 41, 42
—— —— ——, recovery after lumbar puncture in cases of otitic meningitis without injury to, Surg. 160
—— —— (diseases of), demonstration by means of kinematograph (H. C. Thomson), Med. 150
—— —— ——, functional, value of hypnotism in treatment of, Therap. 60
—— ——, effect on action of digitalis, Med. 271
—— —— —— on action of heart, Med. 271, 272, 274
—— ——, instability of, in sufferers from tics, Neur. 43
—— —— of dog suffering from ataxia and involuntary movements, pathological changes found in (F. E. Batten and G. Holmes), Neur. 22
Nettleship, E., pedigree of night-blindness traced by, Epid. 143
Neuralgia, treatment by cocaine ions, Electr. 76
—— of great sciatic nerve, Surg. 167
—— —— —— ——, symptoms, Surg. 168
—— (trigeminal), treatment by excision of Gasserian ganglion (J. Hutchinson, jun.), Clin. 21
—— —— —— by salicylic acid ions, Electr. 76
Neural spindles in ocular muscles, Neur. 73
Neurasthenia with chronic constipation, Med. 147
Neuritis complicating pneumonia, Med. 11
—— of brachial plexus (W. P. Herringham), Clin. 31
—— of great sciatic nerve, Surg. 167
—— —— —— ——, symptoms, Surg. 168
—— and perineuritis of great sciatic nerve, Surg. 167
—— —— —— —— ——, symptoms, Surg. 169
Neuro-fibroma (developing) in case of fibromata of skin (D. Walsh), Derm. 189
——, see also *Macroglossia* neurofibromatosa
Neuro-fibromata (multiple) with spastic paraplegia (T. Thompson), Neur. 63
Neurones, lower, toxic affection of muscles and lower neurones (C. E. Beevor), Neur. 2
Neurotomy (intracranial) unsuccessful in treatment of case of trigeminal neuralgia (J. Hutchinson, jun.), Clin. 21
NEWSHOLME, ARTHUR.—Discussion on etiology and epidemiology of typhoid fever, Epid. 218
——, discussion on Mendelism in relation to disease, Epid. 161
—— —— on plague, Epid. 86
—— —— on preventive medicine at Panama, Epid. 313
——, poverty and disease, as illustrated by the course of typhus fever and phthisis in Irelan (presidential address at opening meeting of Epidemiological Section), Epid. 1
New York, case of chronic "carrier" of typhoid in neighbourhood of, Epid. 211
Nicotine, immunization to, Therap. 36, 38
Nieter and Liefmann, observations on typhoid "carriers" quoted, Epid. 212, 213
Night-blindness, pedigree relating to, illustrating Mendelism, Epid. 143, 163, 164
NITCH, C. A. R.—Case of myositis ossificans, Clin. 107
Nitrogen in urine, Path. 131, 133-143
Nitro-hydrochloric acid, value in dyspepsia, Therap. 20, 21
Nitrous oxide, administration of, production of nystagmus during, Neur. 81
Nocard, criticism of his views respecting relationship between human and avian tuberculosis, Path. 69, 72
Nodular "ringed eruption," case (H. G. Adamson), Derm. 81
Nodule of granuloma annulare, Derm. 142-145
Nodules (subcutaneous rheumatic), associated with advanced pericarditis and endocarditis, Therap. 67, 82, 87
—— (subcutaneous, rheumatic, multiple) (H. French), Clin. 75
—— —— —— ——, in adult (A. E. Garrod), Clin. 13
—— —— —— —— (F. H. Hawkins), Clin. 237

Nodules (symmetrical) on vocal cords (H. Barwell), Laryng. 18

Northcroft, G.—Discussion on classification of dento-facial irregularities, Odont. 144

Nose, accessory sinuses of, radiograph of, defining orbito-ethmoidal cells before operation (StC. Thomson), Laryng. 51

— — —— —— ——, shown by series of skiagrams (P. Watson Williams), Laryng. 121

——, bleeding polypus of (W. H. Kelson), Laryng. 33

——, cyst on floor of (H. J. Davis), Laryng. 97

——, diffuse gummatous infiltration of left half of (W. Jobson Horne), Laryng. 14

——, dryness and crustings of, accompanied by anosmia (C. Fox), Laryng. 13

——, external flattening in case of occlusion of posterior nares (H. Tod), Otol. 133

——, inferior meatus of, great development of, coexistent with feeble development of maxillary sinus, Odont. 22

——, infiltration of cavities of (C. A. Parker), Laryng. 91

——. lupoid ulcer of floor of vestibule of (J. Dundas Grant), Laryng. 110

——, mucocele of anterior ethmoidal cell of left side opened from (D. McKenzie), Laryng. 101

——, pulsating angeioma of (W. D. Harmer), Laryng. 142

——, sarcoma of, case shown after operation (W. Chichele Nourse), Laryng. 55

——. sweating of, in case of intranasal hypertrophy (J. Donelan), Laryng. 47

——, syphilis, case of (H. Tilley), Laryng. 14

——, telangiectases of skin and mucous membranes of (A. Brown Kelly), Laryng. 44

——, ulceration of (W. H. Kelson), Laryng. 63

—— and lip, extensive gummatous ulceration of, with gummatous hepatitis (J. H. Sequeira), Derm. 183

——, pharynx and larynx, lesions in, in case of leprosy (StClair Thomson), Laryng. 74

——, see also *Ala nasi ; Nasal breathing ; Nasal obstruction*

Nostril, right, supposed cyst on floor of, in woman ; specimen (H. J. Davis), Laryng. 139

Nostrils, anterior, case of complete closure of, with partial and progressive atresia of naso-pharynx and oro-pharynx, Laryng. 56

—— (both), case of partial occlusion of, by congenital cutaneous web (G. K. Grimmer), Laryng. 66

——, posterior occlusion, complete, with flattening of nose externally and almost complete absence of nasal septum (H. Tod), Otol. 133

Nourse, W. Chichele.—Bilateral frontal sinusitis after operation, Laryng. 25 ; discussion (p. 26) : Mr. Barwell, Dr. Scanes Spicer, Mr. Nourse (reply)

——, case of epithelioma of the tongue and fauces, shown after operations (pathological report by Wyatt Wingrave), Laryng. 56

—— —— of sarcoma of the nose, shown after operation (with microscopic section) (pathological report by Wyatt Wingrave), Laryng. 55

——, discussion on malignant growth of septum and naso-pharynx removed by external osteo-plastic operation, Laryng. 118

—— —— on case of epithelioma of left tonsil, faucial pillar and uvula, Laryng. 30

—— —— on paresis of right vocal cord, Laryng. 2

—— —— on value of X-rays in sounding and washing out frontal sinus, Laryng. 87

Nucleus, cerebral: tubercle of one sixth nucleus (E. F. Buzzard), Neur. 38

Nutmeg acts as depressant on central nervous system, Therap. 41

——, myristicin active poisonous principle of, Therap. 40, 41, 43

——, oil of, amount required to produce poisonous effect, Therap. 43

—— ——, irritant action, Therap. 42

——, poisoning by (A. R. Cushny), Therap. 39

—— ——, symptoms of, Therap. 39

—— —— ——, due to depressant action on central nervous system, Therap. 41, 42

——, resinous materials in, Therap. 44

Nux vomica in irritable dyspepsia, Therap. 18

Nystagmus, alternate contraction of, Neur. 71, 72

——, connection with tremor, Neur. 81

Nystagmus dependent on disorder of mid-brain centre, Neur. 78
—— in abscess of cerebellum, Otol. 136, 139, 142
—— in unilateral hysterical nerve deafness, Otol. 128, 129
——, lesions giving rise to, situation, Neur. 74
——, mechanism of (Sir W. R. Gowers), Neur. 71
——, problem of, bearing of automatic action of spinal centres on, Neur. 72, 73
——, production of, Neur. 80
—— —— during administration of nitrous oxide, Neur. 81
——, variations in direction of movement, Neur. 78
—— (acquired), in disease, situation of causes, Neur. 76
—— ——, quicker motion in direction of volition feature of, Neur. 77
—— (artificial) production of, Otol. 155
—— (elicited) as test for presence of labyrinthitis, Otol. 71, 80, 82
—— (infantile), Neur. 80
—— ——, connection of reflex action of light with, Neur. 75
—— (miners'), causes of, Neur. 75, 79
—— (spontaneous), rare in labyrinthitis, Otol. 56
—— (thermal), Neur. 82
—— (vestibular or labyrinthine), Neur. 76, 77
—— —— ——, causes of, Neur. 80
—— —— ——, due to deranged impressions from semicircular canals, Neur. 76, 77

Obtundent, paraform as, Odont. 41
Ochronosis (E. Reid), Clin. 57
—— associated with alkaptonuria, Clin. 57, 58
—— —— with carboluria, Clin. 58
Odontological Section, Museum of, specimens added to, described (J. F. Colyer), Odont. 27
Œdema (chronic) of face, case (W. S. Fox), Derm. 13
—— of hands and feet, with mediastinal affection (F. Parkes Weber), Clin. 192
—— (pulmonary, acute suffocative) in case of rheumatic hyperpyrexia, Med. 155
—— —— —— ——, diagnosis from suffocative catarrh of Laennec, Med. 153
—— —— —— ——, with antecedent heart disease, Med. 154
OGILVIE, GEORGE.—Case for diagnosis, Neur. 93, 94
——, Friedreich's disease, Neur. 92
——, hereditary spastic paralysis, Neur. 91; discussion (p. 91): Dr. Guthrie, Dr. Head
——, locomotor ataxia, Neur. 93
——, right hemiplegia following operation, Neur. 94; discussion (p. 95): Sir V. Horsley
OGLE, CYRIL.—Discussion on tumour of mediastinum, Clin. 95
Ogston-Luc operation in treatment of cases of frontal sinus disease (W. S. Syme), Laryng. 99
Old age, see *Warts* (senile)
Olecranon bursa, unusual situation of gouty deposit in (J. G. Forbes), Clin. 53, 80
OLIVER, JAMES.—Discussion on case of uterine fibromyoma during pregnancy, Obst. 77
——, somewhat unique tubal gestation, Obst. 68
Olmer and Terras, on the cutaneous and ophthalmic reactions, alluded to, Path. 76
Omentum, secondary cysts connected with, in case of ovarian dermoid (M. Randall and T. W. P. Lawrence), Obst. 105, 109
OPENSHAW, T. H., C.M.G.—Appliance for obtaining extension of the spine in the treatment of scoliosis and caries, Clin. 196
——, case of congenital absence of the lower part of the tibia, Clin. 8
——, congenital absence of the fibula and outer half of the foot, Clin. 9
——, case of recurrent dislocation of the shoulder cured by operation, Clin. 29; discussion (p. 30): Mr. Jonathan Hutchinson, jun., Mr. Openshaw (reply)
——, discussion on dislocation of patella, with osteo arthritis, Clin. 96
——, multiple dislocations, including congenital dislocation of both hips in child aged 2 weeks, incontestably result of malposition *in utero*, Clin. 106

OPENSHAW, T. H., C.M.G.—Traumatic dislocation of left hip, replaced by manipulation thirteen months after the injury, Clin. 43
Operations under hypnotism, Therap. 57
—— under mesmerism, Therap. 50, 51
Ophthalmic reaction, see *Tuberculin* ophthalmic reaction
Oppenheim, cutaneous tuberculosis inoculated by von Pirquet's method, alluded to, Path. 75
Opsonic index, control of dosage of tuberculin by, Med. 251
—— ——, determinations by, Med. 199, 223
—— —— raised above normal in birds suffering from active tuberculosis, Path. 65
—— ——, relation of human to avian bacillus as tested by means of, Path. 60-65
—— ——, relation to clinical symptoms in pulmonary tuberculosis, Med. 232
—— —— —— to temperature in pulmonary tuberculosis, Med. 224
—— —— —— —— —— —— in febrile cases, Med. 225
—— —— —— —— —— —— in afebrile cases, Med. 228
—— ——, tables of, showing phagocytosis of melanin, Med. 173-187
—— ——, value of, estimation respecting, Med. 238, 239, 251
Opsonin, production *in vitro* by patients' cells in patients' serum, Med. 191-194
Opsonization of melanin, Med. 182
——, strict specificity of, Med. 177, 179, 181
Orbito-ethmoidal cells, definition before operation by means of radiograph of nasal cavities (StClair Thomson), Laryng. 51
—— ——, photograph showing remarkable development of (P. Watson Williams), Laryng. 121
—— ——, stereoscopic slide showing well-marked development of (H. Tilley), Laryng. 123
ORMEROD, J. A.—Case of pityriasis rubra pilaris, Derm. 35; discussion (p. 36): Dr. Pringle
——, congenital pigmentation with atrophic scarring associated with other congenital abnormalities (shown by Dr. H. G. Adamson), Derm. 9
——, discussion on case of extreme lordosis of two years' standing in a woman aged 32, Neur. 58
—— —— on cases of myatonia congenita and of toxic affection of muscles and lower neurones, Neur. 4
—— —— on "tics," Neur. 44
Oro-pharynx and naso-pharynx, partial and progressive atresia of, in case of complete closure of anterior nares (W. Stuart-Low), Laryng. 56
ORTON, G. HARRISON.—Discussion on diagnostic value of Röntgen rays in some diseases of chest, Electr. 46
——, fractures of the scaphoid bone, Electr. 62
——, X-ray diagnosis of renal and ureteral calculi, Electr. 15
Orthodiagraph, diagnostic value of, Electr. 45, 46, 47
—— necessary for measurement of movements of diaphragm, Electr. 47
OSLER, Professor W., F.R.S.—Discussion on case of unusual situation of gouty deposit, Clin. 54
——, discussion on ochronosis, Clin. 57
——, on the association of Raynaud's disease with epileptic fits, quoted, Med. 88
——, on the causation of Raynaud's disease, Med. 88
——, splenic polycythæmia with cyanosis, Clin. 41; discussion (p. 42): Mr. R. G. Haun, Dr. Parkes Weber, Dr. Pasteur, Sir T. Barlow
Ossicles, preservation of, after mastoid operation (A. Logan Turner), Otol. 144, 147
"Ossific diathesis," Path. 106
Osteo-arthritis of elbow, with old fracture of humerus (R. J. Godlee), Clin. 95
—— of hands, with disseminated lupus (A. Whitfield), Derm. 93
—— of knee, with old-standing dislocation of patella (R. J. Godlee), Clin. 96
Osteitis deformans mistaken for sarcomatous growth, Path. 92, 93
——, non-calcifying, plastic, of tibia in adult, how differing from case of hypertrophied callus in infant, Path. 103
Osteoid chondroma, see *Chondroma*, osteoid
Osteoma, difficulty of diagnosis from periosteal sarcoma, Path. 90, 91

Osteoma, diffuse cancellous, of femur following fracture (W. H. Battle and S. G. Shattock), Path. 83-94

—— —— —— —— —— ——, histology, Path. 96

—— —— —— —— —— ——, pathological account and remarks (S. G. Shattock), Path. 94-114

—— —— —— —— —— ——, spontaneous disappearance, Path. 109

Osteotomy, linear, in treatment of coxa valga, Surg. 138

Osteo-myelitis, acute, aural discharge in, Otol. 116

Osteoplastic operation, external, malignant growth of septum and naso-pharynx removed by (P. Watson Williams), Laryng. 115

Otitis rare complication of pneumonia, Med. 11

—— (external, chronic desquamative), aural discharge in, Otol. 116

—— media complicated by meningitis ; see *Meningitis* (otitic)

—— —— (acute) accompanying labyrinthitis, Otol. 69, 82

—— —— (chronic suppurative), cases of, accompanied by labyrinthitis, Otol. 57, 61, 63, 65, 66, 67, 68, 69, 70

—— ——, followed by primary epithelioma (?) of tympanum (A. L. Whitehead), Otol. 34

—— —— (purulent), with deep ulceration of external meatus in case of secondary syphilis (A. Bronner), Otol. 88

Ottweiler, typhoid fever epidemic at, Epid. 209

Outgrowths (bony) from maxilla and mandible (W. H. Kelson), Laryng. 94

Ovariotomy and panhysterectomy in case of sarcomatous ovarian tumour coexisting with carcinoma of uterus (A. H. N. Lewers), Obst. 156

Ovary, cancer of, primary bilateral (T. W. Eden), Obst. 243

—— —— of both ovaries and of sigmoid flexure (J. D. Malcolm), Obst. 236

—— (cysts of, bilateral hæmorrhagic), case of (J. P. Hedley), Obst. 98

—— (cyst of, small), with large hydrosalpinx simulating malignant disease (J. D. Malcolm), Obst. 241

—— (dermoid of), see *Dermoid* (ovarian)

——, encapsulation of sterile fluid in connection with (J. Bland-Sutton), Obst. 229

——, retention of ovaries when possible advocated in treatment of genital tuberculosis, Obst. 261, 263

——, sarcomatous tumour of, coexisting with carcinoma of uterus (A. H. N. Lewers), Obst. 69

—— —— ovarian tumour (perithelioma) coexisting with carcinoma of uterus (A. H. N. Lewers) ; specimen, Obst. 156 ; report of Pathology Committee on, Obst. 158

Ovenden, on supports of pelvic viscera, quoted, Obst. 196

Ovum (human, extremely young ; Teacher-Bryce ovum), demonstration of (J. H. Teacher), Obst. 264

——, position of, in specimen of ovarian associated with intra-uterine pregnancy, Obst. 276

——, relation to corpus luteum in specimen of ovarian associated with intra-uterine pregnancy, Obst. 275

Owen, S. A. Statistics of complications of pneumonia from University College Hospital (1897-1906), Med. 70

Pachydermia laryngis verrucosa et diffusa, specimens showing pathogenesis of (W. Jobson Horne), Laryng. 132

——, syphilitic (StC. Thomson), Laryng. 48

Painblan, on the tuberculin ophthalmic reaction, alluded to, Path. 75

Pain, chief symptom in sciatica, Surg. 168, 169

——, intracranial, source of, Med. 89

—— and spasm in coxa valga, Surg. 135

Palate, functional paresis of palate and cords (E. A. Peters), Laryng. 11

—— (hard), case in which an endothelioma had been removed from (D. McKenzie), Laryng. 62

—— (soft), case of venous angeioma of (H. Tod), Laryng. 73

—— and vocal cords, paralysis of, in case of tabes (C. M. H. Howell), Neur. 66

Panama (City of), insanitation, till recently, notorious, Epid. 306
—— ——, provided with water supply, Epid. 309
—— (Isthmus of), mortality at, historical account, Epid. 303
—— ——, physical features of, Epid. 304
—— ——, progress of preventive medicine at (Sir F. Treves), Epid. 303-313
—— ——, unhealthiness due to yellow fever and malaria, Epid. 304
Panhypertrophy, segmented, case of (S. A. K. Wilson), Neur. 11
Pansinusitis, chronic suppurative, Killian's complete operation performed on right side, and on anterior and inferior walls only on left side (H. Tilley), Laryng. 97
Panhysterectomy (abdominal) in case of difficult labour, Obst. 140
—— for uterine fibroids, showing sarcomatous degeneration, Obst. 151
——, with ovariotomy, in case of sarcomatous ovarian tumour coexisting with carcinoma of uterus (A. H. N. Lewers), Obst. 156
PANTON, P. N.—See *Shattock, S. G.*—Relationship between avian and human tuberculosis
Papilloma of trachea, preparations illustrating (W. Jobson Horne), Laryng. 37
Papillomata, specimen of tonsil composed of mass of (A. R. Tweedie), Laryng. 70
Papules, development of, after inoculating skin with calves' lymph, Path. 74
Paracentesis, performance for ascites twenty-five times in fifteen months; patient well seven years later (F. H. Hawkins), Clin. 190
Paraffinoma, sections of (A. W. Williams), Derm. 23
Paraform, dental uses of (J. E. Spiller), Odont. 41
Parakeratosis variegata (G. Dawson), Derm. 175
—— —— ? case of (H. Radcliffe Crocker and G. Pernet), Derm. 36-38
Paralysis, abducens, crossed, in case of cerebellar abscess (D. R. Paterson), Otol. 88
—— agitans, case (W. Harris), Neur. 98
—— (bulbar), case (H. Batty Shaw), Clin. 55
—— ——, case with unusual position of the head (J. Fletcher Little), Neur. 70
—— of diaphragm and abdominal muscles (permanent) in case of acute anterior poliomyelitis (W. Pasteur), Clin. 51
—— (general, of insane), epileptiform seizures of, Med. 108
—— (infantile), coxa valga associated with, Surg. 125
—— of larynx, case (H. Clayton Fox), Laryng. 107
—— of palate and vocal cords in case of tabes (C. M. H. Howell), Neur. 66
—— (spastic) hereditary, (G. Ogilvie), Neur. 91
—— of tongue, unilateral (W. Jobson Horne), Laryng. 53
—— of vocal cord, first symptom of aneurysm of arch of aorta, Electr. 52
——, see also *Abductor* paralysis, *Adductor* paralysis
Parametria, sheaths of, acts as supports of pelvic viscera, Obst. 45, 198, 200
—— ——, description of, Obst. 54-56
Parametrium, condition of, in case of advanced carcinoma of cervix, Obst. 102
PARAMORE, R. H.—Discussion on the supports of the pelvic viscera, Obst. 59
——, the supports-in-chief of the female pelvic viscera, Obst. 195-214; discussion (pp. 214-228): Professor Paterson, Mr. Arthur Keith, Dr. W. C. Fothergill, Professor Peter Thompson, Dr. Hastings Tweedy, Dr. Briggs, Dr. Alexander Macphail, Dr. Macnaughton Jones, Dr. Inglis Parsons, Dr. Griffith, Dr. Herbert Spencer, Dr. Paramore (reply)
Paraplegia (spastic) with multiple neuro-fibromata (T. Thompson), Neur. 63
—— —— —— (primary), illustrating the nature of Schäfer's reflex (E. Jones), Neur. 59
Parasites, invasion by, simulating tubercular infection in birds, Path. 21
—— (protozoal), diseases caused by, Therap. 22
Parathyroidal adenoma, see *Adenoma*, parathyroidal
Paratyphoid fever, diagnosis of, probable value of intermediate bacillus (P.) in, Path. 81
—— group of bacilli, special characters of, Path. 80
Parents, coöperation of, essential in furthering medical inspection of school children, Epid. 45, 46, 47
Paresis isolated, of right serratus magnus (E. Jones and A. H. Tubby), Neur. 9

Paresis of right vocal cord (D. McKenzie), Laryng. 2
Parey, Ambrose, views on menstruation quoted, Obst. 292
Parker, C. A.—Case of infiltration of the nasal cavities, Laryng. 91; discussion (p. 92): Dr. Pegler, Dr. W. Wingrave, Mr. Parker (reply)
——, case of neoplasm of the right tonsil, Laryng. 140; discussion (p. 141): Mr. C. Baber, Dr. de H. Hall, Dr. L. Lack, Dr. Donelan, Mr. H. Tilley, Dr. D. McKenzie, Mr. Parker (reply)
——, discussion on case of chronic suppuration of maxillary antrum, Laryng. 31
—— and **Pegler, L. H.**—Two cases from which a bleeding polypus of the septum had been removed (with microscopical specimens), Laryng. 26; discussion (p. 28): Dr. StClair Thomson, Dr. Jobson Horne, Dr. Pegler, Mr. Clayton Fox, Dr. J. B. Ball, Dr. Pegler (reply), Mr. Parker (reply)
Parker, Rushton.—Case of severe compound fracture of the arm, Surg. 221
——, discussion on reduction of subcoracoid dislocation of humerus, Surg. 198
——, two cases of ununited fracture; treatment by planting small fragments of bone between the ends, Surg. 232
Parkinson, J. Porter.—Discussion on case of fatal purpura hæmorrhagica, Clin. 123
—— and **Hosford, J. Stroud.**—Cerebellar tumour with proptosis, Clin. 124; discussion (p. 127): Mr. Hosford, Sir T. Barlow, Sir Dyce Duckworth, Dr. Porter Parkinson (reply)
Paronychia, diphtheritic, treatment by copper ions, Electr. 74
Parsons, H. Franklin.—Discussion on etiology and epidemiology of typhoid fever, Epid. 222
Parsons, J. Inglis.—Discussion on the supports of the pelvic viscera, Obst. 222
Partial coupling between hereditary characters, Epid. 153
Pasteur, William.—A case of acute anterior poliomyelitis with permanent paralysis of the diaphragm and abdominal muscles, Clin. 51
——, discussion on complications of pneumonia, Med. 23
—— —— on splenic polycythæmia with cyanosis, Clin. 43
——, statistics of the complications of pneumonia from Middlesex Hospital, Med. 54
Patella, dislocation, ? congenital; operation, cure (D. Drew), Clin. 11
—— —— ——, old-standing, with osteo-arthritis of knee (R. J. Godlee), Clin. 96
Paterson, Professor A. M.—Discussion on the supports of the pelvic viscera, Obst. 214
—— on the relation of the rectum to the other pelvic organs, quoted, Obst. 57
Paterson, D. R.—Crossed abducens paralysis in a case of cerebellar abscess, Otol. 88; discussion (p. 89): Dr. Grant, Mr. Tod, Dr. Paterson (reply)
——, discussion on case of primary epithelioma (?) of tympanum, Otol. 36
—— —— —— of woman aged 27 with almost complete nasal obstruction on left side, with inability to clear nose, Laryng. 140
—— —— on epithelioma of middle ear, ? traumatic, Otol. 92
—— —— on four cases illustrating surgery of sphenoidal sinus, Laryng. 129
—— —— on operative surgery of labyrinthitis, Otol. 79
—— —— on photograph showing remarkable development of orbito-ethmoidal cells, Laryng. 121
—— —— on purulent otitis media with deep ulceration of external meatus in a case of secondary syphilis, Otol. 88
——, unusual case of cerebellar abscess, Otol 23; discussion (p. 23): Mr. A. Cheatle, Mr A. L. Whitehead, Dr. Paterson (reply)
Paterson, Herbert J.—Discussion on removal of goitre by operation, Surg. 79
Paterson, W. B.—Discussion on case of extensive necrosis of mandible, Odont. 32
Pathology, advocacy of broad conception of, Path. 2
Pathology Committee (Obstetrical and Gynæcological Section), report on carcinoma of both ovaries and of sigmoid flexure, Obst. 236
—— ——, report on presumed sarcoma of uterus (considered to be fibromyoma), Obst. 255
—— ——, report on specimen of sarcoma invading myoma, Obst. 156
—— —— —— on sarcoma of cervix, Obst. 155
—— —— —— on sarcoma of uterus, Obst. 248
—— —— —— on specimen of uterine fibroids, Obst. 151
Pawlow, Professor I., researches on digestive system, quoted, Therap. 8, 11, 13

PAYNE, J. F.—Discussion on plague, Epid. 92
PEACOCK, C. N.—Discussion on treatment of children from dental aspect, Odont. 71
PEARSON, S. VERE.—Discussion on vaccine treatment by the mouth, Med. 247
Peas, crossing of, illustrating dihybridism, Epid. 144
——, see also *Sweet peas*
Pectoral muscles, see *Muscles*, pectoral
Pedicle, torsion of, in case of hydrosalpinx (J. P. Hedley), Obst. 95
Pediculosis, greater prevalence among school-girls, Epid. 53
PEGLER, L. HEMINGTON.—Case of tumour of the left vocal cord (œdematous fibroma), Laryng. 8 ; discussion (p. 9), Mr. De Santi, Mr. Robinson, Mr. Barwell, Dr. StClair Thomson, Dr. Clayton Fox, Dr. Pegler (reply)
—— —— —— ——, patient shown after operation, Laryng. 29
——, discussion on bilateral sarcoma of upper maxillæ, Laryng. 22
—— —— on bilateral swelling of the septum nasi, Laryng. 94
—— —— on bony outgrowths from the maxilla and mandible, Laryng. 95
—— —— on case of infiltration of nasal cavities, Laryng. 92
—— —— on effects of long-neglected adenoids on development of upper jaws and nasal septum, Laryng. 86
—— —— on fistulous opening in middle line of neck, Laryng. 4
—— —— on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 105
—— —— on laryngeal disease, Laryng. 120
—— —— on localized pulsating growth of middle turbinate body, Laryng. 1
—— —— on subcutaneous induration of the neck of uncertain origin, Laryng. 113
—— —— on tumour removed from naso-pharynx, Laryng. 10
—— and PARKER, C. A.—Two cases from which a bleeding polypus of the septum had been removed (with microscopical specimen), Laryng. 26 ; discussion (p. 28) : Dr. StClair Thomson, Dr. Jobson Horne, Mr. Clayton Fox, Dr. J. B. Ball, Dr. Pegler (reply)
Pelvic disease in women, treatment by ionic medication in milder cases gratifying, Electr. 136
—— ——, treatment by ionic medication in obstinate cases (S. Sloan), Electr. 126-138
Pelvis : improved demonstration pelvis, with attached fœtal skull for demonstrating mechanism of labour (H. Macnaughton Jones), Obst. 41
——, œdematous fibroma of (W. Tate), Obst. 7
——, retaining ovarian dermoid for two years after obstructing labour (A. Doran), Obst. 35
——, viscera contained in, supports of (W. E. Fothergill), Obst. 43
—— —— —— —— ——, complexity, Obst. 204, 205, 222, 225
——, see also *Diaphragm*, pelvic
Penicillium glaucum, Otol. 116
—— ——, throat organism in chronic aural discharge, Otol. 114, 115
Penis : annular lichen planus affecting penis and right forearm (J. M. H. MacLeod), Derm. 34
——, gonorrhœal warts on, treatment by X-rays (A. D. Reid), Electr. 63
——, ulcer of, with granulomatous tumour of mouth (J. H. Sequeira), Derm. 57
Pepsin, value of, in dyspepsia discussed, Therap. 7, 9, 10, 17
—— —— ——, combined with hydrochloric acid, Therap. 8
—— —— ——, combined with oxalate of barium, Therap. 17, 18
Percussion (cardiac), importance of, Med. 273
Pericarditis complicating pneumonia, Med. 8, 22, 28, 38
——, subcutaneous nodules in rheumatism associated with, Therap. 67, 82, 87
—— and pneumonia complicating infection of lateral sinus (W. Permewan), Otol. 139
Pericardium, post-mortem condition in cases of lymphocythæmia, Clin. 165
Perientheliomata, Path. 168
Perineum, see *Muscles*, perineal, superficial
Perineuritis of great sciatic nerve, Surg. 167
—— —— ——, symptoms, Surg. 169
——, see also *Neuritis* and *Perineuritis*
Perithelioma of ovary, coexisting with carcinoma of uterus, specimen (A. H. N. Lewers) Obst. 156

Perithelioma of ovary, report of Pathology Committee on, Obst. 158

Peritheliomata, Path. 168

Peritoneum, bacteria found in, Path. 108

——, results to, in case of rupture of ovarian dermoids with secondary cysts connected with omentum, Obst. 115

Peritonitis, pneumococcic, complicating pneumonia, Med. 9

——, tuberculous, cases treated by rectal administration of tuberculin and horse serum with success, Med. 204, 205, 206

PERMEWAN, W.—Case of infection of the lateral sinus; pneumonia and pericarditis; ligature of the jugular vein; complete recovery, Otol. 139; discussion (p. 139): Mr. A. L. Whitehead, Dr. Thomas Barr, Dr. Milligan, Mr. Ballance, Mr. Hugh E. Jones, Dr. Kerr Love, Mr. Sydney Scott, Dr. McBride (reply), Dr. Logan Turner (reply), Mr. Henry Wade (reply), Dr. W. Permewan (reply)

PERNET, GEORGE.—Case of pigmentation of the face, Derm. 17

——, discussion on case of (?) dermatitis artefacta, Derm. 85

—— —— —— of ringed eruption on hand, Derm. 65

—— —— on granuloma annulare, Derm. 162

—— —— on lupus erythematosus in a child, Derm. 171

—— —— on mycosis fungoides, Derm. 168

—— —— on scelerodermia (morphœa), Derm. 163

—— and CROCKER, H. RADCLIFFE.—Case for diagnosis, Derm. 36; discussion (p. 38): Dr. J. M. H. MacLeod, Dr. Pringle, Dr. Leslie Roberts

—— ——, case of lichen planus verrucosus treated by violet light, Derm. 38

—— ——, senile warts developing into fungating growths, Derm. 56

—— ——, two cases of elephantiasis græcorum, Derm. 53; discussion (p. 55): Dr. Colcott Fox. Dr. Whitfield, Dr. Radcliffe Crocker, Dr. Rost, Dr. Pringle, Dr. Pernet (reply)

—— ——, unusual case of pustular vegetating dermatitis, with pigmentation changes, in a woman aged 26, Derm. 168; discussion (p. 168): Mr. Hartigan, Dr. Pernet (reply)

"Periodic time" of muscles, Electr. 104

Peroneal atrophy, two cases (W. Harris), Neur. 54

PETERS, E. A.—Functional paresis of the palate and cords in a woman aged 23, Laryng. 11

Petit mal, see *Epilepsy*, minor

Petit, on the tuberculin ophthalmic reaction, alluded to, Path. 75

Pfannenstiel, his case of cerebral hæmorrhage in eclampsia of pregnancy described, Obst. 84

Phagocytosis, amount occurring when human and avian tubercle bacilli are presented to normal human leucocytes in serum of patients suffering from tuberculosis after saturation of serum with human and avian bacillus, Path. 60-65

—— carried out by means of melanin (S. G. Shattock and L. S. Dudgeon), Med. 169-194

——, degrees and limitations under presentation of human and avian tubercle bacilli, Path. 65-69

—— of melanin, see *Melanin*, phagocytosis of

Pharyngeal portion of Eustachian tube, injury to, from operative procedures (W. S. Syme), Otol. 126

Pharynx, Bezold's mastoid empyema discharging into (W. S. Syme), Otol. 125

——, resection of, complete transverse, with laryngectomy for malignant growth of posterior pharyngeal wall (W. S. Handley), Clin. 66

—— and larynx, endothelioma involving lower part of (H. B. Robinson), Laryng. 68

—— ——, extensive adhesions in, of syphilitic origin (H. B. Robinson), Laryng. 67

——, nose, and larynx, lesions in, in case of leprosy (StClair Thomson), Laryng. 74

PHILLIPS, SIDNEY.—Case of fatal acute illness in a child from status lymphaticus, Clin. 75; discussion (p. 79): Dr. Poynton, Dr. Bernstein, Dr. G. A. Sutherland, Dr. A. F. Russell, Dr. Phillips (reply)

——, case of fatal acute ulcerative colitis with some unusual symptoms, Med. 161; discussion (p. 163): Dr. Herringham, Dr. H. H. Tooth, Dr. H. P. Hawkins, Dr. Dalton, Dr. F. Parkes Weber, Dr. A. M. Elliot, Dr. Samuel West, Dr. Gee, Mr. Charters Symonds, Dr. Phillips (reply)

PHILLIPS, SIDNEY.—Case of multiple telangiectases (shown for Dr. Sidney Phillips by Sir F. Semon), Laryng. 44
——, discussion on neuritis of brachial plexus, Clin. 32
——, multiple telangiectases, Clin. 64; discussion (p. 64): Dr. Colcott Fox, Dr. Phillips (reply)
Phlebitis of lateral sinus in case of middle ear suppuration (P. McBride), Otol. 135
Phleboliths causing confusion in X-ray diagnosis of calculi, Electr. 27
Phlebotomy, see *Blood-letting*
Phosphorus in treatment of dyspepsia, Therap. 8
Photographic plate, action of vesical calculi on, in darkness (H. A. Colwell), Path. 127
Phthisis in Ireland, Epid. 36
—— —— contrasted with typhus fever, Epid. 20
—— —— illustrating relation of poverty to disease, Epid. 1
——, irritable dyspepsia of, treatment, Therap. 17
——, see also *Tuberculosis* (pulmonary)
Physostigma and cannabis in treatment of acute stage of chorea, Therap. 77
Pigeons, breeding of, in illustration of predominance of the prepotent as a factor in evolution, Odont. 120
——, feeding of, with tuberculous human sputum, experiments and results, Path. 23
—— —— —— with tuberculous material from birds, experiments and results, Path. 34
——, inoculation with salt suspension of tuberculous lymphatic glands from human subject, Path. 30
—— —— —— with salt suspensions of tubercular organs from other birds, experiments and results, Path. 46
—— —— with tuberculous human sputum, experiments and results, Path. 28
—— —— —— tuberculous organs of guinea-pigs infected by means of human sputum, experiments and results, Path. 31
——, limited pathogenicity of human tubercle bacillus to, Path. 69
Pigmentation changes in unusual case of pustular vegetating dermatitis in woman (H. Radcliffe Crocker and G. Pernet), Derm. 168
—— (congenital), with atrophic scarring, associated with other congenital abnormalities (H. G. Adamson), Derm. 9
—— of face, case (G. Pernet), Derm. 17
——, inheritance of, in silky fowl, Epid. 158
—— in vulval and vaginal tumours indicates high degree of malignancy, Obst. 127
——, irregularity of, in melanotic tumours of vulva, Obst. 126
——, with enlarged spleen and leucopenia, ? splenic anæmia (W. P. Herringham), Clin. 33
—— —— —— —— —— ——, resemblance of case to Addison's disease (W. P. Herringham), Clin. 33
Pike (*Esox lucius*), developmental defects in upper jaw of, specimen showing (A. Hopewell-Smith), Odont. 61
PIRIE, A. HOWARD.—Discussion on electrical treatment of atonic conditions of digestive system, Electr. 109
——, discussion on ionic medication, Electr. 79
——, the electrolytic method of measuring X-rays, Electr. 92; discussion (p. 99): Mr. Deane Butcher, Dr. N. S. Finzi, Dr. Reginald Morton, Dr. W. Ironside Bruce, Dr. A. H. Pirie (reply)
von Pirquet, development of papules after inoculating skin with calves' lymph, alluded to, Path. 74
——, tuberculin cutaneous reaction of, alluded to, Path. 74
Pitts, B., and Shattock, S. G., case of non-calcifying plastic osteitis of tibia, quoted, Path. 102
Pituitary gland, influence on menstruation, Obst. 311
Pityriasis rosea (E. G. Graham Little), Derm. 182
—— rubra pilaris, case (G. W. Dawson), Derm. 13
—— —— —— (J. A. Ormerod), Derm. 35
Pizzini, experimental production of tuberculosis alluded to, Path. 108
Placenta, physiological action of (W. E. Dixon and F. E. Taylor), Obst. 11

Placenta diffusa, case (A. J. M. Routh), Obst. 127
—— praevia, with delivery of complete gestation sac at end of twenty eighth week (T. J. T. McHattie), Obst. 314
Plague endemic at Yunnan, South China, Epid. 92
——, pneumonic, extreme fatality of, Epid. 95
—— ——, origin of, Epid. 94, 95
——, prevention of, by means of inoculation, Epid. 78, 79
—— ——, by measures relating to human species, Epid. 71
—— ——, by measures relating to inanimate objects, Epid. 73
—— ——, by measures relating to lower animals, Epid. 74
—— ——, present methods (W. M. Haffkine), Epid. 71
——, protection of India from invasion by, Epid. 81
——, spread of, by agency of man unimportant, Epid. 71, 72
—— ——, by agency of mice possible, Epid. 93
—— ——, by agency of rats and fleas, Epid. 74, 75, 91, 92, 93
—— ——, deductions regarding, made at Sydney, Epid. 82
Plastering lesion of lupus vulgaris after application of X-rays, Electr. 87
Plates, insertion of, in treatment of pyorrhœa alveolaris, Odont. 62
Pleura, post-mortem condition of, in cases of lymphocythaemia, Clin. 166
Pleural cavities, diagnosis of fluid in, by screen examination, Electr. 41
Pleurisy with effusion in pneumonia, Med. 44
Plimmer, H. G.—Discovery, by experiment, of the destruction of the trypanosome by antimony, alluded to, Therap. 26
Pneumococcus, danger of, Med. 3
——, immunity to effects of, Med. 4
—— in aural discharges, Otol. 107
——, manifestations of infection by, Med. 33, 38, 42, 43, 47, 48
Pneumococci, dead, in treatment of pneumonia, Med. 251
—— and horse serum, oral administration of, in treatment of pneumonia, Med. 211
——, see also *Serum* (antipneumococcic)
Pneumonia, bodily exposure factor in causation of, Med. 35, 41
—— complicated by endocarditis, sudden death from embolism of coronary artery (case referred to in discussion), Clin. 118
—— complications, discussion on, Med. 2; opened by Dr. Hector Mackenzie (p. 2), continued by Mr. A. G. R. Foulerton, Dr. Herringham, Dr. Pasteur; (adjourned discussion, p. 27) Dr. Hadley, Dr. Gossage, Dr. Arthur Latham, Dr. H. A. Caley, Dr. Samuel West, Dr. Tirard, Dr. Fawcett, Dr. Butler Harris, Dr. Langdon Brown, Dr. Hector Mackenzie (reply)
—— ——, distinction between antecedent and resulting, Med. 28
—— ——, in children, Med. 32, 33
—— ——, statistics relating to, compiled at the various London hospitals, Med. 52-71
—— following lupus erythematosus, Med. 23
——, liability of negroes to, Epid. 310, 316
——, presence of knee-jerk in, Med. 36, 41, 42
——, preventive measures against, instituted by Isthmian Canal Commission, Epid. 310
——, ratio of pulse to respiration in, Med. 41
——, spread of, by spitting habits among men, Med. 49
——, treatment by antipneumococcic, antistreptococcic, and antidiphtheritic serum, Med. 36, 41, 47, 51, 54, 61, 65, 67
—— —— by injection of dead pneumococci, Med. 251
—— —— by inoculation, Med. 31
—— —— by oral administration of pneumococci and horse serum, Med. 211
—— —— by serum and vaccines, Med. 15, 16-20
—— —— by vaccine, Med. 45
—— (abdominal), Med. 29, 30

Pneumonia (acute), diffuse congestive stage of, close alliance to suffocative catarrh, Med. 153, 156
—— (cerebral) (so-called), Med. 48
—— (chronic), rarity of, Med. 8
—— and pericarditis complicating infection of lateral sinus (W. Permewan), Otol. 139
——, see also *Plague* (pneumonic)
Pneumothorax treated by aspiration under X-rays (J. Fawcett), Clin. 38
Poisoning from eating shellfish, Path. 79, 82
——, see also *Nutmeg poisoning*
Poliomyelitis, acute anterior, with permanent paralysis of diaphragm and abdominal muscles (W. Pasteur), Clin. 51
Pollard, Bilton, case of hypertrophied callus of tibia and fibula quoted, Path. 101
——, case of localized enlargement of the tibia quoted, Path. 94
Pollock, W. R.—Discussion on menstruation and the calcium metabolism, Obst. 313
Polycythæmia (splenic), analogous to leukæmia, Clin. 41
—— ——, pathology of, Clin. 41
—— ——, vasomotor changes in, Clin. 42, 43
—— ——, symptoms of, Clin, 41, 42, 43
—— ——, with cyanosis (Professor W. Osler, F.R.S.), Clin. 41
—— ——, without cyanosis (R. G. Hann), Clin. 59
Polypharmacy, benefits of, Therap. 24, 26
Polypoid growth (lymphangeioma) of larynx (H. B. Robinson), Laryng. 88
Polypus (bleeding), of nasal septum, removal in two cases (C. A. Parker and L. H. Pegler), Laryng. 26
—— ——, of nose (W. H. Kelson), Laryng. 33
—— (nasal), pathogenesis of certain forms of (P. Watson Williams), Laryng. 52
—— (post-nasal), large, growing from antrum of Highmore simulating naso-pharyngeal fibroma (J. Dundas Grant), Laryng. 102
—— ——, specimen removed from boy (H. Tilley), Laryng. 122
Pons Varolii, hæmorrhage into, as immediate cause of death in eclampsia of pregnancy (N. C. Carver and J. S. Fairbairn), Obst. 79
Poor-law administration in Ireland, Epid. 18
Poorer classes, prevalence of dental caries among, Odont. 71, 72
Population, residual, of Ireland, Epid. 29
Port Swettenham (Federated Malay States), suppression of malaria at, Epid. 315
Porter, W. G.—An unusual sequel to the radical mastoid operation, Otol. 144, 148. [For discussion, see *Barr*, J. Stoddart—Notes on partial grafting . . . in after-treatment of radical mastoid operation]
Potato Famine in Ireland (1847), Epid. 8
Potter, Coubro.—Case of atresia of the naso-pharynx, Laryng. 15; discussion (p. 16): Mr. Cresswell Baber, Mr. De Santi, Dr. Ball, Dr. Jobson Horne
Potter, E. Furniss.—Case of chronic eczema of both auricles and meatuses, with unusual degree of hyperplasia, in a woman aged 43, Otol. 132; discussion (p. 132): Dr. FitzGerald Powell, Dr. Dan McKenzie, Mr. Yearsley, Dr. Kelson
——, discussion on paresis of right vocal cord, Laryng. 2
——, tumour removed from the naso-pharynx of a girl aged 11, fifteen weeks after the operative removal of adenoids, Laryng. 9; discussion (p. 10): Dr. W. H. Kelson, Mr. Robinson, Mr. Rose, Dr. Pegler
Poverty, relation to disease, Epid. 2
—— —— ——, as illustrated by course of typhus fever and phthisis in Ireland (A. Newsholme), Epid. 1
Powders, ante-prandial remedies in dyspepsia should be administered in form of, Therap. 18
Powell, H. FitzGerald.—Discussion on bilateral sarcoma of upper maxillæ, Laryng. 22
——, discussion on cases after the radical maxillary antrum operation, exemplifying simplification of the after-treatment, Laryng. 59
—— —— on case of chronic eczema of both auricles and meatuses, Otol. 132

POWELL, H. FITZGERALD.—Discussion on case of empyema of left frontal sinus, treated by simplified method of Killian's operation, Laryng. 36
——, discussion on case of laryngeal paralysis, Laryng. 107
—— —— —— of lateral sinus disease, Otol. 125
—— —— —— of malignant growth behind cricoid cartilage, Laryng. 111
—— —— —— of mucocele of frontal sinus, Laryng. 71
—— —— —— of tumour of right thyroid lobe, Laryng. 40
—— —— —— of ulceration and destruction of epiglottis, Laryng. 34
—— —— —— of venous angeioma of the soft palate, Laryng. 73
—— —— on effects of long-neglected adenoids on development of upper jaws and nasal septum, Laryng. 85
—— —— on large post-nasal polypus, simulating naso-pharyngeal fibroma, Laryng. 103
—— —— on new instrument for passing a suture through epiglottis, Laryng. 96
—— —— on operative surgery of labyrinthitis, Otol. 77
—— —— on proptosis, due to distension of frontal sinus, Laryng. 51
—— —— on subcutaneous induration of neck of uncertain origin, Laryng. 113
—— —— on subglottic laryngeal tumour Laryng. 91
—— —— on swelling of right arytænoid, Laryng, 108
POWER, F. B.—Discussion on nutmeg poisoning. Therap, 42
POYNTON, F. J.—Discussion on case of methæmoglobinæmia, Clin. 48
——, discussion on case of rupture of brachial plexus at birth, Clin. 219
—— —— —— of unusual situation of gouty deposit, Clin. 54
—— —— on dislocation of patella with osteo-arthritis, Clin. 97
—— —— on fatal illness from status lymphaticus, Clin. 79
—— —— on leucocythæmia, Clin. 178
—— —— on multiple rheumatic nodules in adult, Clin. 14
—— —— on ruptured aortic valve, Clin. 99
PRAUSNITZ, OTTO C. W.—Discussion on etiology and epidemiology of typhoid fever, Epid. 225
Pregnancy, acidosis in (J. B. Leathes). Path. 131
—— (abdominal), myomectomy during (W. C. Swayne). Obst. 129
——. administration of mercury in, value in preventing syphilis in future offspring. Obst. 163. 170, 171
—— complicated by fibroid tumours (F. J. McCann), Obst. 315
—— —— —— ——. saving life of fœtus where possible. Obst. 320
—— —— by large cervical fibroid, subtotal hysterectomy at fourth month (J. Bland-Sutton), Obst. 17
—— —— by large retroperitoneal and subligamentous fibromyoma (J. H. Dauber), Obst. 132
——, eclampsia of, cerebral hæmorrhage as immediate cause of death in. Obst. 92-94
—— —— —— —— into pons Varolii immediate cause of death in (N. C. Carver and J. S. Fairbairn), Obst. 79
——, enucleation of uterine fibromyoma during: premature labour two days later (H Williamson). Obst. 73
——, influence of small-pox epidemic upon, Epid. 278
—— (intra-uterine), death of fœtus occurring in six consecutive pregnancies (H. Williamson and E. L. Holland), Obst. 161-163
—— (ovarian), associated with intra-uterine pregnancy (J. M. Munro-Kerr), Obst. 268
—— —— —— —— ——, microscopic appearances. Obst. 271
—— —— —— —— ——, naked eye description, Obst. 269
—— —— —— —— ——, position of ovum in. Obst. 276
—— —— —— —— ——, relation of ovum to corpus luteum. Obst. 275
—— (tubal), association of salpingitis with, Obst. 252
—— ——, in wall of tubal pus sac (C. Lockyer), Obst. 248
—— ——, rupture of early, causing death within few hours (J. S. Fairbairn), Obst. 57
—— ——, specimens of (W. C. Swayne), Obst. 152
—— ——, unique case (J. Oliver), Obst. 68

Pregnancy, urine in, analyses of, Path. 136, 137
——, vomiting of, see *Vomiting* (severe) of pregnancy
" Presence and absence " hypothesis of relation between alternative pairs of characters, Epid. 148
—— —— —— —— —— —— —— ——, connection with alkaptonuria, Epid. 148, 149
Pressure-forceps, importance of, in enucleation of cystic tumours of suprarenal body, Surg. 217
Pressure-symptoms in case of disease of cervical vertebræ (E. F. Buzzard), Neur. 12
Prickle cells, significance of, in identification of new growths (W. S. Lazarus-Barlow, C. Rowntree), Path. 168, 175
Pringle, J. J.—Case for diagnosis (shown for Dr. E. A Crisp), Derm. 18
——, dermatitis perstans, case for diagnosis, Derm. 18
——, discussion on case for diagnosis, Derm. 38
—— —— —— (factitious ulcers), Derm. 70
—— —— on cases of elephantiasis græcorum, Derm. 56
—— —— on case of annular lichen planus, Derm. 35
—— —— —— of chronic X-ray dermatitis of hands, Derm. 80
—— —— —— of granuloma annulare, Derm. 34
—— —— —— of macular atrophy of scalp, Derm. 60
—— —— —— of pityriasis rubra pilaris, Derm. 36
—— —— —— of ringed eruption, Derm. 48
—— —— —— of varus nodulosus of Brooke, Derm. 86
Pritchard, Urban.—Discussion on case for diagnosis, Otol. 27
——, discussion on case of stenosis of right auditory meatus, Otol. 15
—— —— on deafness resulting from epidemic cerebrospinal meningitis, Otol. 87
—— —— on sudden deafness in case of syphilis, Otol. 9
Proctitis (ulcerative catarrhal) wash-leather colour of mucous membrane in, Surg. 176, 177
——, ulcerative, treatment by colotomy, Surg. 180, 181, 182
—— —— —— by zinc cataphoresis (F. C. Wallis and W. Ironside Bruce), Surg. 176
Proptosis from distension of frontal sinus (StClair Thomson), Laryng. 49
—— —— —— ——, question of operation, Laryng. 50
——, see also *Exophthalmos*
Protozoa, see *Parasites*, protozoal
Prurigo of Hebra (E. G. Graham Little), Derm. 181
Pruritus (long-standing) in woman aged 22 (A. Whitfield), Derm. 22
Pseudo-diphtheria bacillus, presence in cases of otitic meningitis, Surg. 145
Pseudo-pelade of Brocq, see *Folliculitis* decalvans
Pseudo-tubercle bacilli in aural discharges, Otol. 105
Pseudo-xanthoma elasticum of Balzer, case (E. G. Graham Little), Derm. 86
Psoriasis, extensive, with suppurating lesions (J. Galloway), Derm. 41
Pubiotomy (T. Wilson), Obst. 19
——, dangers of, Obst. 23, 24
——, method of delivery after, Obst. 23
—— —— of passing needle in, Obst. 23
——, methods of performance, Obst. 22
——, notes of illustrative case (T. Wilson), Obst. 24-27
——, position of patient's legs during, Obst. 23
——, results of, Obst. 24
——, separation of divided bones in, Obst. 27
——, site for incision in vertex presentations, Obst. 22, 23
Pubo-coccygeus muscle, anatomy and functions, Obst. 207, 210, 220
Pulsating growth in left external auditory meatus (J. B. Ball), Otol. 32
Pulse, ratio of, to respiration in pneumonia, Med. 41
——, slow, see *Bradycardia*
Punnett, R. C.—Mendelism in relation to disease, Epid. 135; discussion (p. 161): Dr. Newsholme, Dr. H. M. Vernon, Dr. A. E. Garrod, Mr. Major Greenwood, jun., Mr. Udny Yule, Dr. T. Lewis, Dr. Fremantle, Sir Shirley F. Murphy, Mr. Punnett (reply)

Purgatives, administration of, should be preceded by use of enema, Med. 146
Purpura, presence in Gaertner infection, Clin. 85
—— hæmorrhagica complicating typhoid fever, Clin. 122
—— ——, death from cerebral hæmorrhage (S. W. Carruthers), Clin. 120
—— ——, treatment by injections of polyvalent antistreptoccic serum, Clin. 123
Purslow, C. E.—Pregnant uterus with fibroid, the latter in a state of "red degeneration," Obst. 42
Pus, passage from antrum to neck without passing through mastoid process (A. H. Cheatle), Otol. 24
Pyo-pericardium cured by drainage (F. J. Steward and A. E. Garrod), Clin. 15
Pyo-pneumothorax, diagnosis determined by Röntgen ray examination, Electr. 12
Pyorrhœa alveolaris, abscess pockets in, Odont. 66, 67
—— ——, age-incidence, Odont. 104
—— ——, connection of gout with, Odont. 104, 105
—— ——, etiology of, Odont. 104
—— —— ——, milk a probable factor, Odont. 106
—— ——, fragility of teeth in, Odont. 105
—— ——, infectivity of, Odont. 105
—— ——, treatment by cataphoresis of diseased roots, Odont. 63, 65
—— —— —— by insertion of plates, Odont. 62
—— —— —— by stapling of teeth (new method), Odont. 63, 65
Pyosalpinx due to traumatism in case of uterine fibroid, Obst. 123
Pyramidal tract degeneration in case of tabes (C. M. H. Howell), Neur. 65
Pyrexia in rubella, Epid. 103

Quinine, combination of, with other drugs, more efficacious than singly against malaria, Therap. 23, 24
——, potency of, in attacking parasite of malaria, Therap. 23
—— —— —— —— —— when limited, Therap. 23
——, small doses of, administration followed by acute scarlatiniform eruption (J. Galloway and Dr. Cohen), Derm. 71
——, Warburg's tincture superior to, in treatment of malaria, Therap. 24
Quinsy, changes in age- and sex-incidence before and during school life, Epid. 57

Rabbits, crossing of, illustrating interdependence of characters, Epid. 145
——, transference of charcoal through intact intestinal mucosa of, Path. 42
——, female, observations and experiments on, respecting relation of metabolism of blood-calcium to menstruation, Obst. 301, 305
Race, influence of, in small-pox epidemic in Trinidad, Epid. 246
Radiography, diagnosis of union of fractured bones by, Surg. 222, 228, 235, 237
Radium, therapeutic applications, Electr. 6
Rainfall, increase of, how far a factor in causation of typhoid fever epidemics, Epid. 173, 174
Ramsden, H. K.—Discussion on treatment of ulcerative proctitis by zinc cataphoresis, Surg. 184
Randall, Martin (introduced by Dr. Herbert Spencer).—Ovarian dermoid with secondary cysts connected with the omentum (with pathological reports and remarks by T. W. P. Lawrence), Obst. 105, 108; discussion (p. 117): Dr. Herbert Spencer, Dr. Galabin, Dr. R. H. Hodgson, Dr. Amand Routh, Dr. Macnaughton Jones
Rangoon, method of treatment of leprosy in, Derm. 56
Ransome, A.—Discussion on rubella, Epid. 125
Rash of rubella, Epid. 102
—— ——, see also *Eruption*
Rats, agency of, in spreading plague, Epid. 74, 75, 91, 92, 93
——, destruction and evacuation of, as means of plague prevention, Epid. 74, 75, 83, 85, 91
——, disappearance of, with removal of garbage, Epid. 87, 88

RAWLING, L. B.—Discussion on fracture through temporal bone, Otol. 11
Raynaud's disease, association with epilepsy, Med. 87, 88
—— —— —— —— with epileptic fits, Med. 88
—— ——, condition of fingers simulating, in case of lupus erythematosus of face (J. H. Sequeira), Derm. 17
—— ——, symptoms resembling, in cases of splenic polycythæmia, Clin. 41, 42
RAYNER, D. C.—Discussion on cerebral hæmorrhage in eclampsia of pregnancy, Obst. 93
Reboul, on etiology of absence of pectoral muscles, alluded to, Clin. 73
von Recklinghausen's disease, case of (Sir M. Morris and W. S. Fox), Derm. 16
Rectum, administration of tuberculin and horse serum by, successful in cases of tuberculous peritonitis, Med. 204, 205, 206
—— —— of tuberculin and saline solution by, diagnostic use, Med. 216
——, douche electrode for introducing current into, Electr. 108
——, inflammation, see *Proctitis*
——, relation of, to other pelvic organs, Obst. 57
——, sarcoma of, wide-seated invasion of bones and lymphatic glands (C. Rowntree), Path. 124
——, ulceration of, treatment by zinc ions, Electr. 75
"Red degeneration" of fibroid in pregnant uterus (C. E. Purslow), Obst. 42
—— —— —— —— complicating pregnancy (F. J. McCann), Obst. 320
"Redundant callus" formation, Path. 107
Reflex (Schäfer's), case of primary spastic paraplegia illustrating nature of (E. Jones), Neur. 59
Reid, Archdall, views on immunity quoted, Odont. 112, 113
REID, A. D.—Case of gonorrheal warts, Electr. 63; discussion (p. 64): Mr. Beddoes, Mr. Deane Butcher
——, discussion on X-ray diagnosis of renal and ureteral calculi, Electr. 30
——, slide of fracture in carpus exhibited, Electr. 62
REID, EDGAR.—Case of ochronosis, Clin. 57; discussion (p. 57): Professor Osler, Dr. A. E. Garrod, Dr. Hale White, Dr. Parkes Weber, Dr. Reid (reply)
RENTON, J. C.—Sciatica and its surgical treatment, Surg. 167; discussion (p. 174): Mr. Warrington Haward, Mr. Swinford Edwards, Dr. Curtis Webb, Dr. Renton (reply)
Reptiles, labyrinth of, stereoscopic photographs illustrating (A. A. Gray), Otol. 164
Resinous materials in nutmeg, Therap. 44
Respiration, ratio of pulse to, in pneumonia, Med. 41
Respiratory failure, relation to suffocative catarrh, Med. 153, 157
Reverse current passing through Röntgen ray tubes, estimation of, Electr. 96
Reversion to type, immunity to dental caries due to, Odont. 116
Rhesus monkey, feeding with avian tubercular material, Path. 53
Rheumatic fever, changes in age- and sex-incidence before and during school life, Epid, 58
—— ——, see also *Rheumatism* (acute)
—— nodules, see *Nodules* (subcutaneous)
Rheumatism, age-incidence of, Therap. 68
—— in childhood (L. G. Guthrie), Therap. 65
——, cutaneous manifestations of, Therap. 67
——, epistaxis in, Therap. 67
——, heart disease following, marked action of digitalis in slowing pulse-rate, Therap. 30
——, micro-organism of, yet unknown, Therap. 66
——, nature of, Therap. 65
——, relation of chorea to, Therap. 71
—— —— ——, shown by history of cases, Therap. 74
——, sore throat in, Therap. 66
—— (acute), treatment by aspirin, Therap. 70, 84, 85
—— —— —— by salicylates, Therap. 69, 83, 84
Rhythmical currents in electro-therapeutics, Electr. 114, 115
Ribs, seventh cervical, with unusual vertebral deformity (E. F. Buzzard), Neur. 69
RICHARDS, H. MEREDITH.—Discussion on medical inspection in schools, Epid. 63

Richards, H. Meredith.—Discussion on rubella, Epid. 128
—— and Brincker, J. A. H.—The potential dangers of water derived from wells in the chalk, Epid. 191, 227
Rickets, diagnosis of, excluded in case of hypertrophied callus of tibia and fibula, Path. 104
—— followed by coxa valga, Surg. 113, 133
Riley, observation of passage of larvæ of *Eristalis* through intestine, Path. 151
Ringed eruption, case of ("lichen annularis," "granuloma annulare"?), Derm. 47
—— —— on hand (G. W. Dawson), Derm. 65
—— ——, nodular, case (H. G. Adamson), Derm. 81
Ringworm, treatment of, Electr. 8
—— —— by Röntgen rays, contra-indicated in children, Electr. 119, 120
—— —— by X-rays; new substance for shielding parts of scalp not to be exposed in (A. Whitfield), Derm. 61
—— of scalp (small-spored), followed by alopecia areata (J. M. H. MacLeod), Derm. 89
——, see also *Horse ringworm*
Robbins, C.—Discussion on treatment of children from dental aspect, Odont. 70
——, discussion on treatment of pyorrhœa, Odont. 65
Roberts, C. Hubert.—A cancerous uterus and glands removed by Wertheim's method, Obst. 61; discussion (p. 64): Dr. Victor Bonney, Dr. Lewers, Mrs. Boyd, Sir Arthur Macan, Dr. Cuthbert Lockyer, Dr. Briggs, Dr. Herbert Spencer
——, discussion on cancer of cervix, treated by high amputation with the cautery, Obst. 35
—— —— on pubiotomy, Obst. 30
Roberts, Leslie.—Discussion on case for diagnosis, Derm. 38
Robinson, H. B.—Case of extensive adhesions in pharynx and larynx of syphilitic origin, Laryng. 67
——, case of polypoid growth (lymphangeioma) of the larynx, Laryng. 88
——, discussion on bony outgrowths from maxilla and mandible, Laryng. 94
—— —— on case of tumour of vocal cord, Laryng. 9
—— —— on cyst on floor of nose, Laryng. 97
—— —— on extensive cellulitis of right side of neck, Laryng. 11
—— —— on tumour removed from naso-pharynx, Laryng. 10
——, endothelioma involving the lower part of the larynx and pharynx, Laryng. 68; discussion (p. 69): Sir F. Semon
Rodent ulcer, case of (J. H. Stowers), Derm. 40
—— ——, multiple (T. J. P. Hartigan), Derm. 8
—— ——, treatment by introduction of zinc ions, Electr. 73; (A. Whitfield), Derm. 21
—— of ala nasi (A. W. Williams), Derm. 62
Röntgenography, plastic, lantern slides illustrating (W. Deane Butcher), Electr. 64
——, see also *Skiagraphy*
Röntgen ray dermatitis, see *Dermatitis*
—— —— tubes, Electr. 18
—— —— ——, estimation of reverse current passing through, Electr. 96
—— —— ——, standardization of, Electr. 94
—— —— ——, transformer of energy, Electr. 93
Rontgen rays, aspiration under, in treatment of pneumothorax (J. Fawcett), Clin. 38
—— —— combined with cauterization in treatment of lupus vulgaris (J. G. Tomkinson), Electr. 83
—— ——, diagnostic value, Electr. 5; see also *Screen examinations*
—— —— —— —— in diseases of chest (A. Stanley Green), Electr. 35
—— —— —— —— —— ——, apparatus, Electr. 36
—— —— —— —— —— ——, discussion on, Electr. 35: Mr. A. Stanley Green, Mr. W. Deane Butcher, Dr. David Arthur, Dr. Halls Dally, Dr. Harrison Orton, Dr. Squire, Dr. Lee, Dr. Lyster, Dr. D. Somerville, Dr. S. West, Dr. Hinds Howell, Dr. Allpress Simmons, Dr. J. A. Codd, Dr. Green (reply)
—— —— —— —— —— ——, importance of erect posture, Electr. 37

Röntgen rays, diagnostic value in diseases of chest, technique, Electr. 37
—— —— —— —— in fracture of carpus (A. D. Reid) Electr. 62
—— —— —— —— —— of scaphoid bone (G. Harrison Orton), Electr. 62; (W. Y. Somerville), Electr. 63
—— —— —— —— in obscure abdominal cases (Professor E. Goldmann), Surg. 14
—— —— —— —— in renal and ureteral calculi, Electr. 15
—— —— —— —— —— —— —— (G. H. Orton), Electr. 15
—— ——, electrolytic method of measuring (H. Pirie), Electr. 92
—— ——, examination of cases of chronic constipation under, Med. 120-147
—— ——, measurement by Sabouraud's pastille, Electr. 93, 94, 95
—— ——, study of method of action enjoined, Electr. 89
—— ——, treatment by, Electr. 7
—— —— —— limitations, Electr. 121
—— —— —— of cancer of stomach, Electr. 120
—— —— —— of endothelioma of lower jaw (W. Ironside Bruce), Electr. 59
—— —— —— of epithelioma of tongue (W. Ironside Bruce), Electr. 59
—— —— —— of exophthalmic goitre (W. Ironside Bruce), Electr. 57, 58
—— —— —— of favus, contraindicated in children, Electr. 119, 120
—— —— —— of leukæmia, Electr. 120, 121
—— —— —— —— (W. Ironside Bruce), Electr. 56, 57
—— —— —— of lupus, Electr. 121
—— —— —— ——, epithelioma following, Electr. 87, 121
—— —— —— of malignant disease, Electr. 120
—— —— —— of mycosis fungoides (H. Radcliffe Crocker), Derm. 75
—— —— —— of ringworm, contraindicated in children, Electr. 119, 120
—— —— —— ——, new substance for shielding parts of scalp not to be exposed in (A. Whitfield), Derm. 61
—— —— —— of sarcoma of abdominal wall (W. Ironside Bruce), Electr. 60
—— —— —— of skin diseases, Electr. 119
—— —— —— of syphilis, Electr. 90, 91
—— —— —— of chronic X-ray dermatitis in measured doses (J. H. Sequeira), Derm. 78
—— ——. value of, in sounding and washing out frontal sinus (StClair Thomson), Laryng. 86
—— ——, variation in output, Electr. 93
—— ——, see also *Dermatitis* (X-ray), *Radiography*, *Röntgenography*, *Skiagraphy*
Rolleston, H. D.—Discussion on vaccine treatment by the mouth, Med. 246
Rombergism as test for presence of labyrinthitis, Otol. 70
Römer, investigations respecting presence of nerve fibres in dentine, quoted, Odont. 49
Rosacea, well-marked, associated with phlyctenular conjunctivitis and ulceration of eyes (E. G. Graham Little), Derm. 181
Rose, F. A.—Discussion on paresis of right vocal cord, Laryng. 3
——, discussion on tumour removed from naso-pharynx, Laryng. 10
——, part of the breast-bone of a chicken which had been impacted in the larynx for nearly forty-eight hours. Laryng. 5; discussion (p. 5): Dr. Donelan, Dr. William Hill, Dr. Jobson Horne, Mr. Rose (reply)
Rosecomb bantams, dominant and recessive characters illustrated by breeding of, Epid. 138
Ross, Dr.—Discussion on cauterization as an adjuvant to radiotherapy. Electr. 90
Ross, Professor Ronald, C.B., F.R.S.—Discussion on preventive medicine at Panama, Epid. 316
Rost, Dr.—Discussion on cases of elephantiasis græcorum, Derm. 56
Rötheln, see *Rubella*
Roughton, J. P.—Lymphangioma of face, Clin. 91; discussion (p. 92): Mr. Godlee, Dr. Parkes Weber, Mr. W. G. Spencer, Dr. Sutherland, Mr. Roughton (reply)
Routh, Amand.—Case of Cæsarean hysterectomy for traumatic atresia of the vagina, the patient having previously undergone a successful operation for a vesico-vaginal fistula due to the same injury, Obst. 1; discussion (p. 7): Dr. Herbert Spencer

ROUTH, AMAND.—Discussion on abdominal hysterotomy for chronic uterine inversion, Obst. 289
——, discussion on case on Caesarean hysterectomy for stenosis of vagina, Obst. 192
—— —— on case of fibroid tumour complicating pregnancy, Obst. 319
—— —— —— of placenta prævia, Obst. 315
—— —— —— of sarcoma of uterus, Obst. 248
—— —— —— of uterine fibro-myoma, Obst. 78
—— —— on cerebral hæmorrhage in eclampsia of pregnancy, Obst. 93
—— —— on intra-uterine death of syphilitic fœtus in six consecutive pregnancies, Obst. 170
—— —— on menstruation and the calcium metabolism, Obst. 313
—— —— on myomectomy during pregnancy, Obst. 136
—— —— on ovarian dermoid with secondary cysts connected with omentum, Obst. 118
—— —— on physiological action of placenta, Obst. 12
—— —— on pregnancy in wall of tubal sac, Obst. 252
—— —— on sarcoma of uterus, Obst. 236, 248
—— —— on the supports of the pelvic viscera, Obst. 58
—— —— on uterine fibroids showing sarcomatous degeneration, Obst. 152
——, placenta diffusa, Obst. 127; discussion (p. 128): Dr. Herbert Spencer
ROWLANDS, R. P.—Excision of body of scapula, Clin. 105
ROWNTREE, CECIL W.—Sarcoma of the rectum in a boy aged 10, Path. 124
——, the significance of prickle cells in the identification of new growths, Path. 175
——, tumour in thigh, Clin. 20
Royal Infirmary, Edinburgh, Electrical Department, work done in (D. Turner), Electr. 118
Rubella (E. W. Goodall), Epid. 101
——, age-incidence in, Epid. 104, 105, 120
—— —— differs from that of measles, Epid. 109
—— and "fourth disease," Epid. 114, 115
——, clinical features of, Epid. 102
——, complications of, Epid. 107
——, diagnosis of, Epid. 128, 129
—— —— by absence of Koplik's spots in, Epid. 111
—— —— from measles, Epid. 108-112
——, etiology of (H. E. Corbin), Epid. 117
——, geographical distribution of, Epid. 104, 118
——, identity of, Epid. 108
—— —— and etiology of (H. E. Corbin), Epid. 117
——, incubation period of, Epid. 103
——, infectivity of, Epid. 105, 119, 127
——, seasonal prevalence of, Epid. 104, 109, 120
—— —— ——, differs from that of measles, Epid. 109
—— symptoms occasionally severe, Epid. 106, 107
Rusca, case of macroglossia neurofibromatosa quoted, Path. 9
RUSHTON, W.—Discussion on classification of dento-facial irregularities, Odont. 144
——, discussion on movements of mandible, Odont. 96
——, discussion on treatment of children from dental aspect, Odont. 71
——, notes on the extraction of an upper wisdom tooth, Odont. 101; discussion (p. 103): Mr. J. G. Turner, Mr. Kenneth Goadby
RUSSELL, A. E.—Discussion on fatal illness from status lymphaticus, Clin. 80
—— on cardiac arrest in idiopathic epilepsy, quoted, Med. 77
——, the pathology of epilepsy, Med. 72; discussion (pp. 112-118): Dr. David Ferrier, Dr. Beevor, Dr. Gossage, Dr. Biernacki, Dr. Lindsay Steven, Dr. Farquhar Buzzard, Dr. A. E. Russell (reply)
"Rut" of animals, pro-œstrum stage of, homologous with menstruation, Obst. 293

Sabouraud's pastille in measurement of Röntgen rays, Electr. 93, 94, 95
Saiki and Inouye, on urine of epileptics, alluded to, Path. 132
St. Bartholomew's Hospital, statistics of complications of pneumonia at (W. Langdon Brown), Med. 55

St. Mary's Hospital, statistics of complications of pneumonia at (1896-1905) (H. A. Caley and F. S. Langmead), Med. 69
St. Thomas's Hospital, statistics of complications of pneumonia at (H. C. Squires), Med. 52
St. Vitus' dance, see *Chorea*
Salicylate of soda combined with bicarbonate of soda and liquor bismuthi, value in rheumatism, Therap. 86
—— —— compared with salicylate of quinine, Therap. 70
Salicylates in treatment of acute rheumatism, Therap. 69, 83, 84, 87
—— —— —— —— ——, advantages and disadvantages compared, Therap. 69, 83, 84, 87
Salicylic acid ions in treatment of trigeminal neuralgia, Electr. 76
Saline solution (normal), oral administration of, with tuberculin (T.R.) and other vaccines (A. Latham, H. D. Spitta and A. C. Inman), Med. 195-239
——, see also *Tuberculin* (T.R.)
Saling, attempt to disprove validity of Levaditi's method of staining *Spirochæta pallida* in sections, Obst. 167, 168, 169
Salpêtrière, School of, in hypnotic teaching, Therap. 55
—— —— —— ——, morbid excitement a feature of, Therap. 64
—— —— —— ——, now defunct, Therap. 63, 64
—— —— —— ——, revived fallacies of mesmerists, Therap. 55
—— —— —— ——, sensational experiments provocative of hysteria, Therap. 64
Salpingitis, association with tubal pregnancy, Obst. 252
——, tuberculous, bilateral, case (F. J. McCann), Obst. 259
SANDILANDS, J. E.—Discussion on medical inspection in schools, Epid. 63
SANDWITH, F. M.—Discussion on plague, Epid. 94
——, discussion on preventive medicine at Panama, Epid. 314
Sarcoid tumours, relation to granuloma annulare, Derm. 150, 151
Sarcoma of abdominal wall, treatment by X-rays (W. Ironside Bruce), Electr. 60
—— arising at site of fracture of humerus, Path. 100
—— of upper air passages, specimens from cases of, and cases simulating (W. Jobson Horne), Laryng. 100
—— of cervix, specimen (Miss L. G. Anderson), Obst. 153
—— ——, report of Pathology Committee on, Obst. 155
——, grape-like, of cervix uteri; removal, with vaginal hysterectomy (F. J. McCann), Obst. 253
—— of femur (H. A. T. Fairbank), Clin. 109
——, recurrent, of femur; excision of parts of femoral artery and vein (C. H. Fagge), Clin. 231
——, bilateral, of upper maxillæ (A. Hutchison), Laryng. 21
—— of nose, case shown after operation (W. Chichele Nourse), Laryng. 55
—— of rectum in boy aged 10; wide-seated invasion of bones and lymphatic glands (C. Rowntree), Path. 124
—— of skin, multiple idiopathic hæmorrhagic (F. Parkes Weber), Clin. 235
—— of uterus, case (C. Lockyer), Obst. 245
—— ——, rarity of, Obst. 233
—— (fibro-myoma) of uterus (J. Bland-Sutton), Obst. 233
—— (periosteal), difficulty of diagnosis of osteoma, subperiosteal hæmatoma, and other lesions from, Path. 90-94
——, see also *Perithelioma*
—— and carcinoma, tumours consisting of, explanation, Path. 170
Sarcomatous ovarian tumour coexisting with carcinoma of uterus (A. H. N. Lewers), Obst. 69
SARGENT, PERCY.—Case of nerve-root grafting, Neur. 7; discussion (p. 8): Dr. H. Head, Dr. Hinds Howell, Dr. Wilson, Dr. Wilfred Harris, Mr. Sargent (reply)
SAVILL, T. D.—Case of lichen plano-pilaris, Derm. 169
——, discussion on arteriosclerosis, Therap. 36
—— —— on case of bromide eruption, Derm. 75
—— —— —— of lupus with unusual features, Derm. 78
—— —— —— of urticaria pigmentosa, Derm. 64

Scalp, favus of, two cases (E. G. Graham Little), Derm. 26
——, lupus erythematosus affecting hands, ears and scalp (J. M. H. MacLeod), Derm. 88
——, macular atrophy of (pseudo-pelade of Brocq) (A. Whitfield), Derm. 60
——, ringworm of (small-spored), followed by alopecia areata (J. M. H. MacLeod), Derm. 89
——, shielding of parts not to be exposed in treatment of ringworm by X-rays, new substance for (A. Whitfield), Derm. 61
Scaphoid bone, congenital absence of both thumbs, trapezium and scaphoid (H. Curtis), Clin. 236
—— ——, fractures of, diagnosis by X-rays (G. Harrison Orton), Electr. 62; (W. G. Somerville), Electr. 63
Scapula, excision of body of (R. P. Rowlands), Clin. 105
Scar, see *Vaccination* scar
Scarlet fever, changes in age- and sex-incidence before and during school life, Epid. 57
—— —— and "fourth disease," Epid. 114, 115
—— ——, see *Eruption* (scarlatiniform)
Scarring (atrophic) in congenital pigmentation, associated with other congenital abnormalities (H. G. Adamson), Derm. 9
SCHELLING, C.—Discussion on classification of dento-facial irregularities, Odont. 144
Schoolboys, greater prevalence of seborrhœa sicca among, Epid. 52
School-children, age-incidence of diseases among, changes in, Epid. 57
—— —— —— ——, table showing, Epid. 56
——, defects in clothing among, Epid. 53
——, medical history card supplied to, Epid. 61, 62
—— —— inspection of, cöoperation of parents and teachers essential, Epid. 45, 46, 47
—— —— —— Gloucestershire scheme (M. Coplans), Epid. 45
—— —— —— remedial measures following, Epid. 53
——, presence of tuberculosis in families of, Epid. 55
——, second attacks of infectious diseases in, Epid. 59
School-girls, greater prevalence of pediculosis among, Epid. 53
Sciatica (J. C. Renton), Surg. 167
——, causes of, Surg. 168
——, diagnosis, Surg. 170
——, forms of, Surg. 167
——, pathology, Surg. 170
——, symptoms of, Surg. 168
——, treatment by hypnotism, Therap. 54
—— —— by static electricity, Surg. 174, 175
—— ——, surgical, Surg. 171
—— —— ——, bloodless methods, Surg. 171
—— —— —— operations, Surg. 171
Sciatic nerve, see *Nerve*, great sciatic
Schäfer's reflex, see *Reflex*
Schaudinn, discovery of *Spirochæta pallida* by, Obst. 164
SCHELLING, Mr.—Discussion on classification of dento-facial irregularities, Odont. 144
Scleroderma, case of (G. A. Sutherland), Clin. 233
—— (morphœa) (H. G. Adamson), Derm. 163
—— and leucodermia, case of (H. G. Adamson), Derm. 43
Scoliosis, treatment of, appliance for obtaining extension of spine in (T. H. Openshaw, C.M.G.), Clin. 196
Scotland, opposition to mesmerism in, Therap. 51
SCOTT, SYDNEY R.—Case of extirpation of the labyrinth, Otol. 5; discussion (p. 6): Dr. W. Milligan, Mr. Scott (reply)
——, discussion on case for diagnosis, Otol. 28
—— —— on fracture through temporal bone, Otol. 12
—— —— on keloid following mastoid operation, Otol. 22

Scott, Sydney R.—Discussion on mechanism of nystagmus, Neur. 81,
——, discussion on sudden deafness in case of syphilis, Otol. 10
—— —— on thrombosis of lateral sinus, Otol. 141
—— —— of vertigo, Otol. 29
—— —— on vestibulotomy, Otol. 154
——, histological preparations of the human cochlea, Otol. 19; discussion (p. 19): Dr. McBride, Mr. A. Cheatle, Mr. Scott (reply)
——, histological preparations of the human labyrinth, Otol. 94; discussion (p. 94): Mr. Cunningham
——, notes of a case of otitic meningitis, with histological specimens (lantern slides) of the labyrinth demonstrating streptococci in situ, Otol. 157
—— and West, C. Ernest.—The operative surgery of labyrinthitis, based upon an experience of thirty cases (with bibliography), Otol. 37-73. [For discussion see under *West*, C. Ernest]
Scraping in treatment of lupus vulgaris, Electr. 84
Screen examinations, Electr. 119
—— ——, dangers, Electr. 119
—— ——, diagnosis of fluid in pleural cavities by, Electr. 41
—— —— in X-ray diagnosis of calculi, Electr. 21
—— ——, method of, Electr. 37
Scrotum, see *Cruro-scrotal region*
Sea-sickness, motor insufficiency of stomach due to, Therap. 11
Seasonal incidence of chorea, Therap. 74
—— —— of rubella, Epid. 104, 109, 120
—— —— of small-pox epidemic in Trinidad, Epid. 246
Seaton, Edward C.—Discussion on the etiology and epidemiology of typhoid (enteric) fever, introductory address, Epid. 169
Seborrhœa, see *Dermatitis* (seborrhoic)
—— sicca, greater prevalence among school-boys, Epid. 52
Segregation of factors representing hereditary characters, Epid. 136
Seheult, R.—On an epidemic of small-pox of irregular type in Trinidad during 1902-4, Epid. 229
Seligmann, C. G., and Shattock, S. G.—An example of incomplete glandular hermaphroditism in the domestic fowl, Path. 3
—— see also *Shattock*, S. G., and others—Relationship between avian and human tuberculosis, Path. 15-73
Semicircular canals, Otol. 41
—— ——, deranged impressions from, causing vestibular or labyrinthine nystagmus, Neur. 76, 77
Semon, Sir Felix.—Case of hysteria with very unusual laryngeal manifestations, Laryng. 133; discussion (p. 135): Dr. J. B. Ball
—— case of multiple telangiectases (for Dr. Sidney Phillips), Laryng. 44
—— —— probable combination of tuberculosis and syphilis in the larynx, Laryng. 135; discussion (p. 136): Dr. Watson Williams, Dr. StClair Thomson, Dr. de Havilland Hall, Mr. De Santi, Sir F. Semon (reply)
——, discussion on case of complete closure of the anterior nares, with partial and progressive atresia of the naso-pharynx and oro-pharynx, Laryng. 57
——, discussion on case of congestion of right vocal cord, Laryng. 18
—— —— —— of functional aphonia, Laryng. 80
—— —— —— —— —— in a male, Laryng. 77, 78
—— —— —— of a girl aged 17, with a genuine baritone voice, Laryng. 61
—— —— —— of laryngeal disease, Laryng. 120
—— —— —— of intrinsic laryngeal neoplasm in a man aged 75, Laryng. 82
—— —— —— of laryngeal ulceration, Laryng. 23
—— —— on cases of multiple telangiectases, Laryng. 46
—— —— on case of partial occlusion of both anterior nares by a congenital cutaneous web, Laryng. 67

Semon, Sir Felix.—Discussion on case of symmetrical nodules on cords, Laryng. 19
——, discussion on case of tumour of right thyroid lobe, Laryng. 39, 40
—— —— —— of ulceration and destruction of epiglottis, Laryng. 34
—— —— on endothelioma involving lower part of larynx and pharynx, Laryng. 69
—— —— on malignant disease of the larynx, Laryng. 131
—— —— on new instrument for intralaryngeal operations, Laryng. 25
—— —— on specimen of subglottic hyperplasia with considerable narrowing of tracheal airway, Laryng. 125
——, note on epiglottis holders, Laryng. 41
Sepsis, oral, prevention of, by removal of deciduous molars, Odont. 38, 39
Septic diseases, value of urotropine in, Obst. 118
Septum (nasal), absence, almost complete, in case of complete occlusion of posterior nares (H. Tod), Otol. 133
—— ——, bilateral swelling of (P. De Santi), Laryng. 93
—— ——, bleeding polypus, removal in two cases (C. A. Parker and L. H. Pegler), Laryng. 26
—— ——, deflection of, extreme form (H. Barwell), Laryng. 5
—— in phthisical patient (A. H. Burt), Laryng. 111
—— —— ——, marked (StClair Thomson), Laryng. 130
——, suppurating hæmatoma of, with fistulous opening in gingivo-labial recess (D. McKenzie), Laryng. 92
—— ——, and naso-pharynx, malignant growth of, removed by external osteoplastic operation (P. Watson Williams), Laryng. 115
—— ——, and upper jaws, effects of long-neglected adenoids on development of (J. Donelan), Laryng. 84
Sequeira, J. H.—Case for diagnosis, Derm. 57
——, case of chronic X-ray dermatitis of the hands; removal of warts by measured doses of the X-rays, Derm. 78; discussion (p. 80): Sir Malcolm Morris, Dr. Radcliffe Crocker, Dr. Whitfield, Dr. J. M. H. MacLeod, Dr. Pringle
——, case of granulomatous swellings at left angle of mouth and in right inguinal region, Derm. 92
—— —— of lupus with unusual features suggesting lupus pernio, Derm. 76; discussion (p. 78): Dr. Galloway, Dr. Radcliffe Crocker, Dr. Whitfield, Dr. Savill
—— —— of morphœa, Derm. 90
—— —— of tuberculides, Derm. 39
——, lupus erythematosus of the face with a condition of the fingers simulating Raynaud's disease, Derm. 17
——, two cases of congenital syphilis, with late cutaneous and mucous membrane lesions of the gummatous or phagedænic type, Derm. 183
Sequestra in labyrinthitis, Otol. 53, 67
Sequestrotomy in labyrinthitis, Otol. 68
Serratus magnus (right), isolated paresis of (E. Jones and A. H. Tubby), Neur. 9
Serum: amount of phagocytosis occurring when human and avian tubercle bacilli are presented to normal human leucocytes in serum of patients suffering from tuberculosis after saturation of serum with human and avian bacillus, Path. 60-65
—— of birds, inhibitive action upon leucocytes of normal human blood, Path. 68
——, injection of, in treatment of pneumonia, Med. 15, 16-20
——, see also *Horse* serum
—— (antidiphtheritic, fresh), hypodermic administration increases coagulability of blood, Med. 198
—— —— —— —— —— —— —— in treatment of broncho-pneumonia, Med. 197
—— —— in treatment of arthritis; recovery, Med. 197
—— —— —— of pneumonia, Med. 36, 67
—— (antimeningococcic), value of, Surg. 164
—— (antipneumococcic) in treatment of pneumonia, Med. 36, 11, 51, 54, 65, 67
—— —— —— ——, reasons for former failure, Med. 47

Serum (antistreptococcic) in treatment of pernicious anæmia, Med. 197
—— —— —— —— —— of pneumonia, Med. 41, 61
—— —— —— —— —— of streptococcic infection, Med. 197
—— —— (polyvalent), injections in treatment of purpura hæmorrhagica, Clin. 123
—— (immune), amounts of phagocytosis of melanin on presentation of melanin to washed cells of patients and to washed normal cells in immune serum, Med. 184-191
—— ——, de-opsonization by saturation with melanin, Med. 177, 179, 181
—— ——, heating of, causing reduction of phagocytosis towards suspension of melanin, Med. 182
—— ——, immune cells must work in, to form correct estimate of patient's hæmophagocytic resistance, Med. 190, 191, 194
—— ——, presentation of melanin to washed leucocytes in; increase of phagocytosis of melanin as compared with normal serum, Med. 173-178
—— ——, reduction of phagocytosis of normal cells by saturation of immune serum with melanin against bacterium, or suspension of melanin, or against both, Med. 179, 181
—— (Marmorek's anti-tuberculous), value of, Med. 198
—— (normal), result of presentation of melanin to washed leucocytes of immune blood in, Med. 183, 184
Serum-disease, occurrence of, Med. 237
Serum-rash, cases of, Med. 202, 203, 249
Serum-reactions of patient suffering from bone abscess due to paratyphoid infection, Path. 81
Sex-incidence of diseases among school children, changes in, Epid. 57
—— of small-pox epidemic in Trinidad, Epid. 247
Sex-limited diseases, Epid. 153
Sexual characters, secondary, of fowls, influence of testis upon (C. E. Walker), Path. 153
—— gland in birds, indications of hermaphroditic condition of, Path. 7
Sharp, J. Gordon.—Discussion on rheumatism and chorea in childhood, Therap. 85
Shattock, S. G.—Case of chondrifying sarcoma of humerus following upon a fracture, quoted, Path. 100
——, presidential address at opening meeting of Pathological Section, Path. 1
——, three specimens of the larvæ of *Eristalis tenax* passed by the bowel, Path. 147
—— and Battle, W. H.—A remarkable case of diffuse cancellous osteoma of the femur following a fracture, in which similar growths afterwards developed in connection with other bones, Path. 83-114
—— and Dudgeon, Leonard S.—Observations upon phagocytosis carried out by means of melanin, more especially with reference to the part taken by the leucocytes in infectious diseases, Med. 169-194
—— and Pitts, B., case of non-calcifying plastic osteitis of tibia, quoted, Path. 102
—— and Seligmann, C. G.—An example of incomplete glandular hermaphroditism in the domestic fowl, Path. 3
—— —— ——, Dudgeon, L. S., and Panton, P. N.—A contribution to the study of the relationship between avian and human tuberculosis, Path. 15-73
—— and Spencer, W. G.—A case of macroglossia neurofibromatosa, Path. 8
Shaw, H. Batty.—Case of bulbar paralysis, Clin. 55
——, hepato-splenomegaly with ascites, Clin. 56
—— and Bradford, J. Rose, cases of lymphocythæmia quoted, Clin. 131
Sheaths of parametria as supports of pelvic viscera, Obst. 45, 198, 200
—— ——, description of, Obst. 54-56
Sheep, inheritance of horns in, crossing experiments to investigate, Epid. 154, 155
——, Southdown, specimens showing normal and hypertrophied pharyngeal tonsils in (H. J. Davis), Laryng. 138
Shellfish, poisoning from eating, Path. 79, 82
—— —— ——, comparable to meat poisoning by *Bacillus enteritidis* Gaertner, Path. 82
Shenton, E. W. H. Discussion on X-ray diagnosis of renal and ureteral calculi, Electr. 32

Sherlock, E. B.—Discussion on hypnotism, Therap. 63
Sherrington, C. S., F.R.S., discovery of automatic action of spinal centres by, Neur. 72
Shillitoe, A.—Case of circinate erythematous syphilide, Derm. 21
Shock as an agent in causation of chorea compared with traumatism or chill, Therap. 72
Shoulders, recurrent dislocation of, cured by operation (T. H. Openshaw, C.M.G.), Clin. 29
——, see also *Humerus*
Sigmoid flexure: carcinoma of both ovaries and of sigmoid flexure (J. D. Malcolm), Obst. 236
Sigmoid sinus, left, thrombosis of, with right temporo-sphenoidal abscess; macroscopic and microscopic preparations from case (A. Logan Turner and H. Wade), Otol. 138
—— ——, thrombosis of, operation (A. Logan Turner), Otol. 137
Silky fowl, inheritance of pigmentation in, Epid. 158
Silver nitrate, applications of, in cases of ulcerative proctitis, Surg. 179, 183, 184, 185, 186
—— ——, application to teeth in treatment of dental caries, Odont. 39
—— —— method (Levaditi's) of staining *Spirochæta pallida* in sections, Obst. 164, 165
—— —— —— —— —— —— ——, description, Obst. 166
Simmons, G. Allpress.—Discussion on diagnostic value of Röntgen rays in some diseases of the chest, Electr. 52
——, discussion on X-ray diagnosis of renal and ureteral calculi, Electr. 34
——, exhibition of colour photographs taken on the Lumière autochrome plate, Electr. 34
Simpson, Professor W. J.—Discussion on plague, Epid. 88
Sington, Harold S. (introduced by Dr. Handfield-Jones).—Tumour of fœtal head, Obst. 158; with pathological report by Dr. Reginald Miller (p. 160); discussion (p. 160): Dr. Herbert Spencer
——, tumour of fœtal head exhibited, Obst. 104
Sinus, see *Ethmoidal*, *Frontal*, *Lateral*, *Sigmoid*, *Sphenoidal Sinus*
Sinusoidal currents in treatment of atonic conditions of digestive system, Electr. 106, 107
Skiagraphy, carefulness in, important in coxa vara, Surg. 113, 136
——, progress in, Electr. 1
Skin, development of papules on, after inoculation with calves' lymph, Path. 74
——, diseases of, treatment by Röntgen rays, Electr. 119
——, fibromata of, with developing neurofibroma (D. Walsh), Derm. 189
——, leiomyoma of (multiple) (J. M. H. MacLeod), Derm. 50
——, multiple hereditary developmental angiomata (telangiectases) of skin and mucous membranes (F. Parkes Weber), Clin. 65
——, sarcoma of, multiple, idiopathic hæmorrhagic (F. Parkes Weber), Clin. 235
—— tumours, multiple, of doubtful nature (H. A. Lediard), Clin. 18
——, inflammation of, see *Dermatitis*
——, see also *Dermatology*; *Tuberculosis*, cutaneous
Skull (fœtal) attached to improved demonstration pelvis for demonstrating mechanism of labour (H. Macnaughton Jones), Obst. 41
——, post-mortem appearances of, in case of congenital defect of muscular system, Path. 158
——, see also *Cranium*
Sleep, cessation of tics during, Neur. 43
——, electrical production of, Electr. 11, 106
—— and post-epileptic coma, Med. 99
Sleeping sickness, comparative value of drugs used against, Therap. 26
—— ——, see also *Trypanosomes*
Sloan, Samuel.—Ionic medication in the treatment of some obstinate cases of pelvic disease in women, Electr. 126
Small-pox, account of prevalence in prevaccination times, Epid. 229
——, definition of, Epid. 242
——, diagnosis of, difficulties in, Epid. 239-241
—— epidemic in Trinidad during 1902-4, Epid. 229
—— —— ——, age-incidence, Epid. 242
—— —— ——, attacks on recently vaccinated, Epid. 284

Small-pox epidemic in Trinidad, comparison of vaccinated and unvaccinated cases in respect to liability of attack, Epid. 280
—— —— ——, complications in cases, Epid. 273
—— —— ——, convalescence of cases, Epid. 272
—— —— ——, desiccation in cases, Epid. 268
—— —— ——, eruptive period, Epid. 252
—— —— ——, hæmorrhagic case, Epid. 282
—— —— ——, infectivity slight, Epid. 236
—— —— ——, influence of, in pregnancy, Epid. 278
—— —— ——, initial rashes, Epid. 252
—— —— ——, influence of race upon, Epid. 246
—— —— —— —— of season upon, Epid. 247
—— —— ——, maturation in cases, Epid. 257
—— —— ——, peculiar confluent form of, Epid. 283
—— —— ——, sex-incidence of, Epid. 246
—— —— ——, treatment of cases, Epid. 272
—— —— ——, vaccination of children born of variolous mothers, Epid. 293
—— —— —— ——, results of, performed during desquamation or soon after recovery from disease, Epid. 289
——. prevalence in villages of Venezuela, 1902, Epid. 231, 232
——, susceptibility of negro race to, Epid. 246
——, variations in virulence of epidemics and mortality-rate, Epid. 295
Smith, A. Lionel.—Discussion on cerebral hæmorrhage in eclampsia of pregnancy, Obst. 92
Smith, F. J.—Discussion on growth of malignant disease, Surg. 19
Smith, Heywood.—Discussion on abdominal hysterotomy for chronic uterine inversion, Obst. 289
—— —— on case of caseating carcinoma of corpus uteri, Obst. 121
Smith, Travers.—Discussion on hairball in the stomach, Clin. 215
Smurthwaite, H.—Discussion on bilateral sarcoma of upper maxillæ, Laryng. 22
—— —— on case of functional aphonia, Laryng. 79
—— —— —— of laryngeal ulceration, Laryng. 24
—— —— —— of nasal obstruction, Laryng. 76
Social and sanitary conditions of Ireland, Epid. 22
Sodium bicarbonate, in dyspepsia, Therap. 6, 10
—— ——, combined with salicylate of soda and liquor bismuthi, value in rheumatism, Therap. 86
Somatic cells, variations in, Odont. 110
Somerville, D.—Discussion on diagnostic value of Röntgen rays in some diseases of the chest, Electr. 51
Somerville, W. Y.—Fracture of scaphoid, Electr. 63
Soper, G. A., account of typhoid "carrier," quoted, Epid. 211
Soper, William.—Discussion on therapeutics of indigestion, Therap. 19
——, the reminiscences of an apprentice fifty years ago, Therap. 32
Sore throat in rheumatism, Therap. 66
Sorgo and Suez on the causation of endocarditis by tubercle bacilli, alluded to, Path. 121
Spanton, W. D.—Discussion on primary unilateral tuberculosis of Fallopian tube, Obst. 187
Spasms, differential diagnosis of tics from, Neur. 46, 47
Speech, tics of, Neur. 44
Spencer, Herbert R.—Cancer of cervix in a woman aged 26 treated by high amputation with the cautery, Obst. 31; discussion (p. 35): Dr. C. H. Roberts, Dr. Spencer (reply)
——, caseating carcinoma of the corpus uteri, Obst. 119; discussion (p. 121): Dr. Heywood Smith, Dr. T. G. Stevens, Dr. Herbert Spencer (reply)
——, discussion on abdominal hysterotomy for chronic uterine inversion, Obst. 290
—— —— on bilateral tuberculous salpingitis, Obst. 263
—— —— on case of Cæsarean hysterectomy for traumatic atresia of vagina, Obst. 7

SPENCER, HERBERT R.—Discussion on case of Cæsarean hysterectomy for stenosis of vagina, Obst. 192
——, discussion on case of chorionepithelioma of uterus, Obst. 17
—— —— —— of difficult labour, Obst. 149
—— —— —— of fibroid tumour complicating pregnancy, Obst. 320
—— —— —— of hæmatosalpinx complicating myoma, Obst. 124
—— —— —— of paravaginal fibromyoma, Obst. 322
—— —— —— of placenta diffusa, Obst. 128
—— —— —— —— —— prævia, Obst. 315
—— —— —— of pregnancy complicated by large cervical fibroid, Obst. 18
—— —— —— of sarcoma of cervix, Obst. 155
—— —— —— of tubercle of cervix uteri, Obst. 67
—— —— —— of tumour of fœtal head, Obst. 160
—— —— —— of uterine fibro-myoma during pregnancy, Obst. 77
—— —— on cerebral hæmorrhage in eclampsia of pregnancy, Obst. 92
—— —— on creatinin excretion in lying-in women, Obst. 194
—— —— on development of early human ovum, Obst. 277
—— —— on hydrosalpinx and ovarian cyst simulating malignant disease, Obst. 242
—— —— on intra-uterine death of syphilitic fœtus in six consecutive pregnancies, Obst. 170
—— —— on myomectomy during pregnancy, Obst. 135
—— —— on ovarian dermoid with secondary cysts connected with omentum, Obst. 117
—— —— on physiological action of placenta, Obst. 11
—— —— on pubiotomy, Obst. 29
—— —— on removal of cancerous uterus and glands by Wertheim's method, Obst. 66
—— —— on sarcoma of uterus, Obst. 236
—— —— on the supports of the pelvic viscera, Obst. 60, 225
—— —— on uterine fibroids showing sarcomatous degeneration, Obst. 152
—— —— on Wertheim's operation for carcinoma of cervix, Obst. 104
——, ovarian dermoid with secondary cysts connected with the omentum. (Martin Randall, introduced by Dr. Herbert Spencer), Obst. 105
——, ovarian tumour with a foot of adherent small intestine successfully removed from a patient aged 58, Obst. 172
SPENCER, WALTER G.—Description of disease of bones in goats, quoted, Path. 104
——, lateral curvature rapidly developing in a boy, Clin. 105
——, discussion on lymphangioma of face, Clin. 92
—— —— on removal of goitre by operation, Surg. 77
——, reduction of an old subcoracoid dislocation of the humerus by excavating the glenoid cavity through a posterior intermuscular incision, Surg. 187; discussion (p. 197): Mr. Warrington Haward, Mr. Rushton Parker, Mr. Thomas H. Kellock, Mr. Spencer (reply)
—— and SHATTOCK, S. G.—A case of macroglossia neurofibromatosa, Path. 8
Sphenoidal sinus: bilateral empyema of frontal, ethmoidal and sphenoidal sinuses: operative treatment, case showing result (H. Tilley), Clin. 196
—— ——, surgery of, cases illustrating (StClair Thomson), Laryng. 127
—— ——, X-ray photograph showing Hajek's hook in (H. J. Davis), Laryng. 135
SPICER, FREDERICK.—Laryngeal disease in a man aged 40, Laryng. 11; discussion (p. 12): Dr. Scanes Spicer, Mr. De Santi, Dr. W. Hill, Dr. F. Spicer (reply)
SPICER, R. H. SCANES.—Case of laryngeal disease for diagnosis, Laryng. 12
——, case of intrinsic laryngeal neoplasm in a man aged 75, Laryng. 81; discussion (p. 82): Sir F. Semon, Dr. McBride
—— —— intrinsic neoplasm of the left vocal cord in a man aged 76, Laryng. 80
——, discussion on abscess in left cerebral frontal lobe, originating from suppuration in left frontal sinus, Laryng. 125
—— —— on bilateral sarcoma of upper maxillæ, Laryng. 22
—— —— on case of bilateral frontal sinusitis, Laryng. 26
—— —— —— of functional aphonia, Laryng. 79

Spicer, R. H. Scanes.—Discussion on case of laryngeal disease, Laryng. 12
——, discussion on case of mucocele of frontal sinus, Laryng. 72
—— —— —— of partial occlusion of both anterior nares by a congenital cutaneous web, Laryng. 67
—— —— on effects of long-neglected adenoids on development of the upper jaws and nasal septum, Laryng. 85
—— —— on extreme form of septal deflection, Laryng. 6
—— —— on fistulous opening in middle line of neck, Laryng. 3
—— —— on localized pulsating growth of middle turbinate body, Laryng. 1
—— —— on new instrument for passing a suture through epiglottis, Laryng. 96
Spiller, J. E.—The dental uses of paraform, Odont. 41; discussion (p. 42): Mr. H. Baldwin, Mr. William Hern, Mr. Gabell, Mr. A. E. Baker, Mr. E. B. Dowsett, Mr. J. E. Spiller (reply)
Spinal animal, explanation of term, Neur. 72
Spinal cord, centres of, automatic action of, bearing on problem of nystagmus, Neur. 72, 73
—— —— —— —— —— ——, due to loss of volition, Neur, 72, 73
—— —— —— —— development of alternate contraction of opposing muscles by, Neur. 72
—— ——, sclerosis of, treatment by electrolysis, Electr. 123
—— ——, secondary growths on, in case of tumour of choroid plexus of fourth ventricle, Neur. 28
Spine, caries of, with clubbing of fingers and toes (P. M. Heath), Clin. 234
—— (extension of), appliance for obtaining, in treatment of scoliosis and caries (T. H. Openshaw, C.M.G.), Clin. 196
——, lateral curvature of, rapidly developing in a boy (W. G. Spencer), Clin. 105
Spirochæta fœtida throat organism in chronic aural discharge, Otol. 115
—— *pallida*, description of, Obst. 164
—— ——, discovery of, Obst. 164
—— ——, evidence of being specific organism of syphilis, Obst. 164-169
—— —— —— —— —— —— —— shown by constant presence in organs of congenitally syphilitic fœtuses, Obst. 167
—— ——, presence of, in fœtal tissues (H. Williamson and E. L. Holland), Obst. 161-163
—— ——, sections and film preparations showing, exhibited (Mr. Kettle), Derm. 80
—— ——, staining in films by Giemsa's stain, Obst. 164, 165, 167, 168
—— —— —— in sections by Levaditi's silver nitrate method, Obst. 164, 165, 166
—— *pertenuis*, Obst. 165
—— *refringens*, description of, Obst. 165
Spirochætæ in aural discharges, Otol. 109, 111, 121
Spitta, H. D., see *Latham*, A.—Preliminary communication on the administration of tuberculin (T.R.) and other vaccines by the mouth
Spitting habits, see *Expectoration*
Spleen, enlargement of, in cases of lymphocythæmia, Clin. 160
—— ——, with polycythæmia and cyanosis (Professor W. Osler), Clin. 41
—— —— —— without cyanosis (R. G. Hann), Clin. 59
—— —— and leucopenia accompanying pigmentation (W. P. Herringham), Clin. 33
——, post-mortem condition in cases of lymphocythæmia, Clin. 166
Splints, application of, in ununited fractures, Surg. 237
—— —— ——, in treatment of fractured humerus, Surg. 230, 231
Spratling on blood-pressure in epilepsy, quoted, Med. 83
Spriggs, E. I.—Discussion on termination of nerves in teeth of mammals, Odont. 58
Sputum, tuberculous human, feeding of pigeons with, experiments and results, Path. 23
—— —— ——, inoculation of pigeons with, experiments and results, Path. 28
—— —— —— —— with tuberculous organs of guinea-pigs infected by means of, Path. 31
Squames, acid-fast, in chronic desquamative external otitis, Otol. 116
—— ——, old non-nucleated, in aural discharges, Otol. 99, 121
—— —— —— —— ——, mistaken for tubercle bacilli, Otol. 99

SQUIRE, J. EDWARD, C.B.—Discussion on diagnostic value of Röntgen rays in some diseases of chest, Electr. 48

SQUIRES, H. C.—Statistics of the complications of pneumonia from St. Thomas's Hospital, Med. 52

Staining method of Bethe described, Odont. 50

—— processes used for aural discharges, Otol. 98

Stammering distinct from tics, Neur. 44

Staphylococci, oral administration, dosage, Med. 223

—— —— —— ——, effect of, Med. 219

—— —— —— —— with horse serum, Med. 200, 201, 202, 203

——, varieties of, in aural discharges, Otol. 112

Staphylococcic infection, treatment by oral administration of staphylococci together with horse serum, Med. 200-202

—— ——, see also under *Tuberculous* infection

Staphylococcus, presence in cases of otitic meningitis, Surg. 145

Staphylococcus albus, presence in viscera, Path. 108, 109

Stapling of teeth in treatment of pyorrhœa alveolaris, Odont. 63-65

Starvation diet, high coefficient of ammonia in urine under, Path. 139

State medicine, Mendelism in relation to, Epid. 165

Status epilepticus, Med. 106

—— lymphaticus, fatal acute illness in child due to (S. Phillips), Clin. 75

Stenosis of right auditory meatus (W. H. Kelson), Otol. 14

—— of vagina, three cases of Cæsarean hysterectomy for (K. Das), Obst. 188

STEVEN, J. LINDSAY.—Discussion on pathology of epilepsy, Med. 116

STEVENS, T. G.—Discussion on case of caseating carcinoma of corpus uteri, Obst. 121

STEWARD, F. J.—Case of mucocele of the frontal sinus, Laryng. 71; discussion (p. 71): Mr. H. Tilley, Dr. D. Grant, Dr. F. Powell, Dr. S. Spicer, Mr. Stuart-Low, Dr. Milligan, Mr. Waggett, Dr. J. B. Ball, Mr. Steward (reply)

——, discussion on case of unilateral paralysis of tongue, Laryng. 54

—— —— —— of venous angeioma of the soft palate, Laryng. 73

—— and GARROD, A. E.—Case of pyo-pericardium cured by drainage, Clin. 15; discussion (p. 16): Dr. S. West, Mr. Godlee

STEWART, PURVES.—Discussion on cerebral gummata, Neur. 20

——, discussion on condition of nervous system of ataxic dog, Neur. 26

—— —— on unilateral hysterical nerve deafness, Otol. 128, 129

—— —— on unilateral nerve deafness, Otol. 130

—— —— on wasting of the small muscles of the hand in a girl aged 20, and her father aged 46, Neur. 64

——, gross lesion of post-central gyrus, associated with astereognosis, Clin. 220

STEWART, T. GRAINGER.—Discussion on case of disease of cervical vertebræ with pressure symptoms, Neur. 14

——, note upon the symptomatology of tumours growing in the fourth ventricle, Neur. 26; discussion (p. 36): Dr. Ferrier, Dr. F. E. Batten, Dr. Wilson, Dr. C. E. Beevor, Dr. Grainger Stewart (reply)

Stigmata present in unilateral hysterical nerve deafness, Otol. 127, 128, 129

STILL, G. F.—Hairball in the stomach, Clin. 212; discussion (p. 214): Mr. C. A. Ballance, Dr. Dalton, Dr. A. Masters, Dr. Travers Smith, Mr. T. Sutton Townsend, Dr. Still (reply)

Stokes-Adams disease (heart-block), Med. 73

Stomach, cancer of, treatment by Röntgen rays, Electr. 120

——, dilatation of, acute, following pneumonia, Med. 24

—— ——, combined with dyspepsia of central nervous origin, treatment, Therap. 8

—— ——, complicating pneumonia, Med. 32

——, functions of, Therap. 13

——, hairball in; removal (G. F. Still), Clin. 212

——, hypersensitive condition of, Therap. 14

Stomach, motor insufficiency of, Therap. 6, 11
—— —— ——, due to chill, Therap. 11
—— —— ——, in sea-sickness, Therap. 11
—— —— ——, treatment, Therap. 6
—— motor spasm of, treatment, Therap. 6
—— organic disease of, functional dyspepsia due to, Therap. 9
—— "over-educated," of gourmets, Therap. 14
Stowers, J. H.—Case of infective granuloma of septic origin, Derm. 7
—— —— —— —— ——, report on, Derm. 32
——, case of lupus erythematosus in a child, Derm. 170; discussion (p. 171): Dr. Crocker, Dr. Pernet, Dr. Stowers (reply)
—— —— of molluscum contagiosum in an adult, Derm. 172
—— —— of rodent ulcer, Derm. 40
——, discussion on multiple tumours of skin, Clin. 20
Streptococci, oral administration, effect of, Med. 219
—— —— —— —— ——, with horse serum, Med. 202, 203
——, varieties of, in aural discharges, Otol. 111, 114
Streptococcic infection, improvement under administration of antistreptococcic serum, Med. 197
—— ——, see also under *Tuberculous* infection
Streptococcus, histological specimens of labyrinth (lantern slides), demonstrating streptococci in situ from case of otitic meningitis (S. R. Scott), Otol. 157
——, presence in case of otitic meningitis, Surg. 145, 158
——, see also *Serum* (antistreptococcic)
Streptothricial organisms in aural discharges, Otol. 105, 112
Stridor, laryngeal, congenital, case (J. Dundas Grant), Laryng. 110
Strychnine in treatment of dyspepsia, Therap. 8
Stuart-Low, W.—Case after operation for angina Ludovici, Laryng. 130; discussion (p. 130): Dr. P. McBride, Mr. C. Fox. Dr. J. B. Ball, Mr. Stuart-Low (reply)
——, case of complete closure of the anterior nares, with partial and progressive atresia of the naso-pharynx and oro-pharynx, Laryng. 56; pathological report (W. Wingrave) (p. 57); discussion (p. 57): Mr. Baber, Sir Felix Semon, Dr. StClair Thomson, Mr. Stuart-Low (reply)
——, cases after radical maxillary antrum operation, exemplifying simplification of the after-treatment, Laryng. 58; discussion (p. 58): Dr. J. B. Ball, Mr. H. Tilley, Dr. Jobson Horne, Dr. F. Powell, Dr. McBride, Mr. C. Fox, Mr. Stuart-Low (reply)
——, discussion on abscess in left cerebral frontal lobe, originating from suppuration in left frontal sinus, Laryng. 126
—— —— on case of chronic empyema of left frontal sinus, treated by simplified method of Killian's operation, Laryng. 36
—— —— —— —— suppurative pansinusitis in which Killian's complete operation was performed on the right side and on the anterior and inferior walls only on the left side, Laryng. 97
—— —— case of gumma of larynx, Laryng. 20
—— —— —— of mucocele of frontal sinus, Laryng. 72
—— —— —— of nasal syphilis, Laryng. 14
—— —— on extreme form of septal deflection, Laryng. 6
—— —— on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 104
—— —— on malignant growth of septum and naso-pharynx removed by external osteoplastic operation, Laryng. 118
—— —— on new instrument for intralaryngeal operations, Laryng. 24
—— —— —— —— for passing a suture through epiglottis, Laryng. 96
—— —— on proptosis due to distension of frontal sinus, Laryng. 50
—— —— on vaccine treatment by the mouth, Med. 248
Sturridge, Ernest.—Case demonstrating a point in the treatment of pyorrhœa, Odont. 62; discussion (p. 65): Mr. Robbins, Mr. Badcock, Mr. W. J. May, Dr. Kirk Davenport, Mr. W. Hern, Mr. H. Baldwin, Mr. Sturridge (reply)

Sturridge, Ernest.—Discussion on treatment of children from dental aspect, Odont. 76
Sudan, ravages of malaria in, Epid. 314
Suez and Sorgo, on the causation of endocarditis by tubercle bacilli, quoted, Path. 121
Sugar, enormous increase in production under modern civilization, Therap. 48
——, moderate consumption advocated, Therap. 48
—— (invert), cause of dental caries, Odont. 34, 35
Sulphæmoglobinæmia, intracorpuscular; case of cyanotic anæmia referred to Committee concluded to be one of, Clin. 197, 198
Suppurating lesions present in case of extensive psoriasis (J. Galloway), Derm. 41
Suprarenal body, cystic tumour of, bronzing of skin in, Surg. 214
—— —— —— ——, diagnosis, Surg. 214, 215
—— —— —— ——, history of injury in cases, Surg. 208, 209, 213, 214
—— —— —— ——, pain in, Surg. 202, 207-211, 214
—— —— —— ——, report of cases recorded, Surg. 201-213, 218
—— —— —— ——, surgical pathology, Surg. 213
—— —— —— ——, symptoms, Surg. 214
—— —— —— ——, treatment by incision and drainage, Surg. 209, 215
—— —— —— —— ——, removal by enucleation, Surg. 202, 209, 210, 211, 212, 215, 216
—— —— —— —— —— —— ——, importance of pressure-forceps in, Surg. 217
Suprarenal capsules, cirrhosis of, found post mortem in case of leucodermia with symptoms of Addison's disease (N. Dalton), Clin. 209
Surgery, see *Operations*, and under names of organs and regions
Surrey, county of, typhoid fever in, causes of prevalence various, Epid. 171, 172, 173
Sutherland, G. A.—Discussion on congenital absence of pectoral muscles, Clin. 74
——, discussion on fatal illness from status lymphaticus, Clin. 79
—— —— on lymphangioma of face, Clin. 92
—— —— on treatment of pneumothorax with aspiration under X-rays, Clin. 39
——, scleroderma, Clin. 233
Suture, new instrument for passing it through epiglottis (C. Horsford), Laryng. 95
Swan, R. H. Jocelyn.—Discussion on transverse resection of pharynx with laryngectomy for malignant growth of pharynx, Clin. 69
Swayne, W. C.—Abdominal myomectomy during pregnancy, Obst. 129. [For discussion see under *Dauber*, J. H.—Case of pregnancy complicated with large retroperitoneal and subligamentous fibromyoma]
——, exhibition of two microscopic sections from carcinoma of uterus showing cell changes, Obst. 153
——, specimens of tubal gestation, Obst. 152
——, uterine fibroids showing sarcomatous degeneration (with report of Pathology Committee), Obst. 151; discussion (p. 152): Dr. Herbert Spencer, Dr. Amand Routh, Dr. Cuthbert Lockyer, Dr. Macnaughton Jones, Dr. Swayne (reply)
Sweating of nose in case of intranasal hypertrophy (J. Donelan), Laryng. 47
Sweet, Edwin, remarks on performance of arterio venous anastomosis, quoted, Clin. 206
Sweet-peas crossing of, illustrating interaction of characters, Epid. 149
——, gametic coupling of characters in, Epid. 151
Sweetstuffs, consumption of, cause of dental caries, Odont. 35, 70, 71, 72, 73
Swimmers, proportion among school children, Epid. 51
Swine fever, outbreaks of, simultaneous occurrence on same premises with typhoid outbreak, Epid. 185
Sycosis (coccogenic), case of (T. C. Fox), Derm. 11
Sydney, deductions regarding spread of plague made at, Epid. 82
Syme, W. S.—Case of Bezold's mastoid empyema which discharged into the pharynx, Otol. 125
——, discussion on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 105
—— —— on mastoid operation, Otol. 146
—— —— subcutaneous induration of neck of uncertain origin, Laryng. 113

SYME, W. S.—Discussion on vestibulotomy, Otol. 156
——, injury to pharyngeal portion of Eustachian tube from operative procedures, Otol. 126; discussion (p. 126): Dr. Milligan, Dr. McBride, Dr. Syme (reply)
——, lateral sinus disease, operation; cure, Otol. 123; discussion (p. 125): Mr. A. Cheatle, Dr. FitzGerald Powell
——, series of stereoscopic photographs of patients operated on by various methods for disease of the frontal sinus, Laryng. 99
SYMONDS, C. J.—Case showing the phonetic condition after removal of one vocal cord, Clin. 185; discussion (p. 185): Mr. Sampson Handley, Mr. Symonds (reply)
——, case three years and nine months after complete excision of the larynx, showing an improved method of speaking, Clin. 184
——, discussion on acute ulcerative colitis, Med. 166
Symphysiotomy, Obst. 20
Syncope, attacks of, Med. 80
—— ——, passing into minor epilepsy, Med. 80
—— and convulsive seizures, association of, with bradycardia and other cardiac affections, Med. 73
Synovitis of knee-joint, tuberculous, arthrectomy twice performed (D. Drew), Clin. 10
Syphilide, circinate erythematous (A. Shillitoe), Derm. 21
Syphilis cause of extensive adhesions in pharynx and larynx (H. B. Robinson), Laryng. 67
—— cause of intra-uterine death of fœtus, Obst. 161
—— —— —— —— ——, proved by demonstration of presence of *Spirochæta pallida* in tissues, Obst. 162
——, evidence for *Spirochæta pallida* being specific organism of, Obst. 164-169
——, pachydermia probably due to (StC. Thomson), Laryng. 48
—— probably combined with tuberculosis in larynx (Sir F. Semon), Laryng. 135
——, specimens from case of (A. Whitfield), Derm. 172
——, treatment by Röntgen rays, question discussed, Electr. 90, 91
—— (cerebral), see *Gummata*, cerebral
—— (congenital), diagnosis of, excluded in case of hypertrophied callus of tibia and fibula, Path. 104
—— ——, two cases of, with late cutaneous and mucous membrane lesions of gummatous or phagedænic type (J. H. Sequeira), Derm. 183
—— (nasal), case of (H. Tilley), Laryng. 14
—— (secondary), purulent otitis media with deep ulceration of external meatus in case of (A. Bronner), Otol. 88
—— (tertiary) probable cause of aortitis in case of angina pectoris, Clin. 116, 117, 118
—— ——, sudden deafness occurring under treatment for (R. Lake), Otol. 7
Syringomelia (H. G. Turney), Neur. 89
Syrphidæ, family of (including genus *Eristalis*), close resemblance of many species to humble-bees and wasps explains ancient belief regarding origin of bees, Path. 151

Tabes dorsalis, cause of chronic constipation, Med. 135
—— —— with paralysis of palate and vocal cords (C. M. H. Howell), Neur. 66
—— —— with pyramidal tract degeneration (C. M. H. Howell), Neur. 65
—— mesenterica, treatment by raw meat-juice, Med. 246
Talipes equino-varus (congenital), association with congenital defect of muscular system (R. Howard), Path. 157, 165
Tandler and Halban, views on function of levator ani muscle, quoted, Obst. 213
—— ——, views of, on pelvic visceral supports, quoted, Obst. 204
TATE, W. W. H.—Discussion on pregnancy in wall of tubal pus sac, Obst. 252
——, œdematous fibroma of pelvis, Obst. 7; discussion (p. 10): Mr. Alban Doran
Taxis, immediate, in treatment of chronic uterine inversion, Obst. 282, 283, 285
TAYLOR, FRANK E.—Discussion on abdominal hysterotomy for chronic uterine inversion, Obst. 289

TAYLOR, FRANK E., and DIXON, W. E.—An epidiascopic demonstration on "the physiological action of the placenta," Obst. 11. [For discussion see under *Dixon*, W. E.]

Taylor, Frederick, case of lymphocythæmia, quoted, Clin. 131

——, views on etiology of lymphocythæmia, quoted, Clin. 131, 170

TAYLOR, JAMES.—Case of myopathy, Neur. 7

——, discussion on case of disease of cervical vertebræ with pressure symptoms, Neur. 14

TAYLOR, JAMES.—On X-ray diagnosis of renal and ureteral calculi, Electr. 34

TAYLOR, SEYMOUR.—Discussion on dislocation of patella with osteo-arthritis, Clin. 97

——, ruptured aortic valve, Clin. 97; discussion (p. 98): Sir John Broadbent, Dr. F. J. Poynton, Dr. Theodore Williams, Dr. Parkes Weber, Dr. Seymour Taylor (reply)

Tea, weak, permissible to dyspeptics, Therap. 15

TEACHER, JOHN H.—Demonstration of an extremely young human ovum (Teacher-Bryce ovum). Obst. 264. [For discussion see under *Kerr*, J. M. Munro—Ovarian pregnancy associated with an intra-uterine pregnancy]

Teachers, coöperation of, essential in furthering medical inspection of school children, Epid. 45, 46, 47

Teeth, bad development of, connected with ingestion of cooked vegetable and farinaceous food, Therap. 48

——, cavities of, testing for acid, Odont. 119, 123

——, cleanliness of, natural, Odont. 117, 118

—— —— —— and artificial, Odont. 115, 116

——, cleansing of (artificial cleanliness), Otol. 117, 118

—— ——, in prevention of dental caries, Odont. 33, 34, 37, 39, 72

——, diseases of, in children, treatment (J. F. Colyer), Odont. 33, 72

——, extraction of, under hypnotism, Therap. 57

——, fragility of, in pyorrhœa alveolaris, Odont. 105

—— (individual), irregularities of, not related to dento-facial irregularities, Odont. 142, 143

—— of fossil fishes (J. Humphreys), Odont. 7

—— of mammalia, termination of nerves in (W. J. Law), Odont. 45

——, relation to one another, determination by position of first molar, Odont. 143

——, roots of, diseased, in pyorrhœa alveolaris, treatment by cataphoresis, Odont. 63, 65, 67

——, stapling of, in treatment of pyorrhœa alveolaris, Odont. 62-65

——, structure of, in relation to dental caries, Odont. 36

——, supernumerary, case of (R. McKay), Odont 129.

——, see also *Caries* (dental), *Molar*, *Wisdom tooth*

Telangiectases (bilateral) of trunk, with history of epistaxis in childhood and recent rectal hæmorrhage (T. Colcott Fox) Derm. 66

——, multiple, case of (S. Phillips) Clin. 64

—— —— —— (Sir F. Semon for S. Phillips), Laryng. 44

—— —— —— (E. B. Waggett), Laryng. 70

—— —— hereditary developmental (angiomata), of skin and mucous membranes with recurring epistaxis (F. Parkes Weber), Clin. 65

—— —— —— ——, of skin and mucous membranes, associated with recurring hæmorrhages (F. Parkes Weber), Laryng. 43

—— —— of skin and mucous membranes of nose and throat (A. Brown Kelly), Laryng. 44

—— of feet and legs (spreading) (Sir M. Morris and S. E. Dore) Derm. 89

Telangiectasis of cheek (H. G. Adamson), Derm. 44

Temperature, guide to dosage of tuberculin (T.R.), Med. 220, 221, 222

—— in cases of lymphocythæmia, Clin. 160

—— —— pulmonary tuberculosis, relation of opsonic index to, Med. 224

——, increase of, in pulmonary tuberculosis, relation to exercise, Med. 249, 251

—— (inverse) of miliary tuberculosis, effect of tuberculin in, Med. 214

Temporal bone, fracture through, specimens illustrating (A. Cheatle), Otol. 10

—— ——, specimens of, illustrating operations on the labyrinth (C. E. West), Otol. 94

—— —— (right), specimen showing how pus may reach neck without passing through mastoid process (A. H. Cheatle), Otol. 24

Temporo-sphenoidal lobe (left), abscess of, occurring in course of recent middle-ear suppuration; patient exhibited after operation (A. Logan Turner), Otol. 137

—— —— (right), abscess of, with left sigmoid sinus thrombosis, macroscopic and microscopic preparations from case (A. Logan Turner and H. Wade), Otol. 138

Terras and Olmer, on the cutaneous and ophthalmic reaction, referred to, Path. 76

Testicle, chorionepithelioma of, histological discovery of, Path. 1

——, influence of, upon secondary sexual characters of fowls (C. E. Walker), Path. 153

Tetanization of heart, Med. 254

Therapeutical Society, brief account of history of (T. E. Burton-Brown), Therap. 1

Thermal sensation, delayed, in case of muscular atrophy (Mr. Kilgour for Dr. L. Guthrie), Neur. 10

Thigh, see *Femur*

Thomas, H. O. (of Liverpool), allusion to his skilful management of fractures, Surg. 229

Thompson, H. Theodore.—Multiple neurofibromata and spastic paraplegia, Neur. 63; discussion (p. 63): Mr. D. Armour, Dr. Head (reply)

——, wasting of the small muscles of the hand in a girl aged 20, and her father aged 46, Neur. 63; discussion (p. 64): Dr. H. Howell, Dr. P. Stewart, Dr. Head (reply)

Thompson, J. Ashburton.—Protection of India from invasion by bubonic plague, Epid. 81; discussion (pp. 86-100), including discussion on Mr. Haffkine's paper: Dr. Newsholme, Major Arnim, Professor W. J. Simpson, Dr. J. F. Payne, Col. Macpherson, Dr. Sandwith, Dr. Fremantle, Dr. C. J. Martin, F.R.S., Dr. Chalmers, Mr. Haffkine (reply), Dr. Ashburton Thompson (reply)

Thompson, Professor Peter.—Discussion on the supports of the pelvic viscera, Obst. 220

—— on superficial perineal muscles, quoted, Obst. 48

—— on the levator ani, quoted, Obst. 54

—— on the supports of the pelvic viscera, quoted, Obst. 54

Thomson, Professor Arthur, on influence of jaw growth on cranial form, alluded to, Odont. 23

Thomson, H. Campbell.—Case of extreme lordosis of two years standing in a woman aged 32, Neur. 57; discussion (p. 58): Dr. W. Harris, Dr. Ormerod, Dr. F. Buzzard

——, case of tumour of the motor area of the brain, which was operated on, Neur. 96; discussion (p. 97): Dr. Guthrie, Dr. G. Holmes

——, demonstration of the use of the kinematograph in medicine, Med. 150

——, discussion on cases of myatonia congenita and of toxic affection of muscles and lower neurones, Neur. 4

——, hemiplegia with involuntary movements, Neur. 55, 95; discussion (p. 56): Dr. E. Jones, Dr. Beevor, Dr. G. Holmes, Dr. Batten (reply), Dr. C. Thomson (reply)

——, intracranial tumour, Neur. 96

Thomson, StClair.—Case of leprosy with lesions in the nose, pharynx and larynx, Laryng. 74

——, case of marked deviation of the septum in a man, Laryng. 130

—— —— of proptosis from distension of the frontal sinus, Laryng. 49; discussion (p. 50): Mr. Herbert Tilley, Dr. Lambert Lack, Dr. W. Hill, Mr. Stuart-Low, Mr. Barwell, Dr. FitzGerald Powell, Dr. StClair Thomson (reply)

—— —— of syphilitic pachydermia, Laryng. 48; discussion (p. 48): Dr. Graham Forbes, Mr. Clayton Fox, Dr. StClair Thomson (reply)

——, discussion on bleeding polypus of septum, Laryng. 28

—— —— on case of chronic suppuration of maxillary antrum, Laryng. 32

—— —— —— of complete closure of the anterior nares with partial and progressive atresia of the naso-pharynx and oro-pharynx, Laryng. 57

—— —— —— of lupus of epiglottis, Laryng. 107

—— —— —— of tumour of vocal cord, Laryng. 9

—— —— on extreme form of septal deflection, Laryng. 6

—— —— on fistulous opening in middle line of neck, Laryng. 4

—— —— on large post-nasal polypus simulating naso-pharyngeal fibroma, Laryng. 104

—— —— on new instrument for intralaryngeal operations, Laryng. 25

—— —— on paresis of right vocal cord, Laryng. 3

THOMSON, STCLAIR.—Discussion on specimen of tonsil apparently composed of mass of papillomata, Laryng. 70
——, discussion on swelling of right arytænoid, Laryng. 108
—— —— on tuberculosis and syphilis in larynx, Laryng. 136
——, extensive tuberculosis of the larynx in a woman aged 40, completely cicatrized after treatment with the galvano-cautery and tracheotomy, Laryng. 9; discussion (p. 9): Dr. Dundas Grant
——, four cases illustrating the surgery of the sphenoidal sinus, Laryng. 127; discussion (p. 129): Dr. J. B. Ball, Dr. Paterson, Dr. W. Williams, Dr. StClair Thomson (reply)
——, radiograph of the nasal cavities, Laryng. 51; discussion (p. 52): Dr. Watson Williams
——, results of double frontal sinus operation, Laryng. 13
——, two radiographs to illustrate the value of the X-rays in sounding and washing out the frontal sinus, Laryng. 86; discussion: Mr. H. Tilley, Dr. Westmacott, Mr. C. Nourse, Dr. W. Williams, Mr. C. Fox
——, CARGILL, L. V., and TURNER, W.—Case of abscess in the left cerebral frontal lobe, originating from suppuration in the left frontal sinus, Laryng. 125. [For discussion see *Cargill*, L. V.]
Thorax, description of, in case of congenital defect of muscular system, Path. 158
THORNE, ATWOOD.—Discussion on localized pulsating growth of middle turbinate body, Laryng. 2
Throat, telangiectases of skin and mucous membranes of (A. Brown Kelly), Laryng. 44
"Throat organisms" in chronic aural discharge, Otol. 115
Thrombosis complicating pneumonia, Med. 14
—— of lateral sinus followed by meningitis serosa, recovery (H. Tod), Otol. 30
—— —— ——, leucocytosis in, Otol. 137, 138, 139, 142
—— of sigmoid sinus; operation (A. Logan Turner), Otol. 137
—— of left sigmoid sinus with right temporo-sphenoidal abscess; macroscopic and microscopic preparations from case (A. Logan Turner and H. Wade), Otol. 138
Thumb: congenital absence of both thumbs, with trapezium and scaphoid (H. Curtis), Clin. 236
Thursfield, J. H., case of macroglossia neurofibromatosa, quoted, Path. 8
Thymus gland as primary seat of disease in acute lymphocythæmia, theory respecting, Clin. 170
—— ——, enlargement of, with status lymphaticus, Clin. 77, 78
—— ——, hypertrophy of, complicating goitre, Surg. 78
Thyroid gland, atrophy of, association with myxœdema, importance of discovery, Path. 1
—— ——, malignant disease of, operation results unsatisfactory, Surg. 36
—— ——, secretion from, deficiency in, not connected with arteriosclerosis, Therap. 34
—— ——, tumour of right lobe of (J. Donelan), Laryng. 39
—— —— —— —— ——, specimen and microscopic sections (J. Donelan), Laryng. 46
—— and thymus glands, involvement in avian tuberculosis, Path. 18
Thyrotomy for tuberculous laryngitis, case exhibited one year and nine months after operation (H. Barwell), Laryng. 7
Tibia, congenital absence of lower part (T. H. Openshaw, C.M.G.), Clin. 8
——, non-calcifying plastic osteitis of, Path. 102
—— and fibula, hypertrophied callus of, Path. 101
Tics, absence of original external stimulus a qualification of, Neur. 42
——, cessation of, during sleep, Neur. 43
——, confusion with chorea, Therap. 71
——, definition of, Neur. 41, 42
—— develop occasionally from chorea, Neur. 46
——, differential diagnosis from hysterical manifestations, Neur. 44
—— —— —— —— from spasms, Neur. 46
——, discussion on (opened by Dr. Henry Head, continued by Dr. Ormerod, Dr. James Collier, Dr. Wilfred Harris, Dr. Farquhar Buzzard, Dr. Leonard Guthrie), Neur. 41-48

Tics, instability of nervous system in sufferers from, Neur. 43

——, movements of, differ from those of chorea, Neur. 43

—— of habit, Neur. 42

—— of speech, Neur. 44

—— of volition, Neur. 42, 43

——, so-called spasmodic torticollis, instance of, Neur. 44, 45

——, stammering distinct from, Neur. 44

——, treatment and control of, Neur. 47, 48

Tiegel, experiments respecting transport of bacteria to viscera, alluded to, Path. 108

TILLEY, HERBERT.—Case of "bridle" formation in the larynx, Laryng. 16

——, case of chronic empyema of left frontal sinus operated upon two years ago by a simplified method of Killian's operation, Laryng. 35; discussion (p. 36): Dr. William Hill, Mr. Stuart-Low, Mr. Barwell, Dr. FitzGerald Powell, Mr. Herbert Tilley (reply)

—— —— chronic suppurative pansinusitis in which Killian's complete operation was performed on the right side and on the anterior and inferior walls only on the left side, Laryng. 97; discussion (p. 98): Mr. Stuart-Low, Mr. Tilley (reply)

—— —— of congestion of the right vocal cord, Laryng. 18; discussion (p. 18): Sir Felix Semon, Dr. Ball, Mr. Tilley (reply)

—— —— of epithelioma of the left vocal cord, Laryng. 60

—— —— of fibrous constriction of the trachea, Laryng. 35; discussion (p. 35): Dr. Ball, Mr. Tilley (reply)

—— —— of keloid following operation for acute mastoid suppuration, Otol. 20; discussion (p. 21): Dr. Milligan, Mr. West, Dr. Donelan, Mr. Fagge, Mr. Waggett, Dr. Jobson Horne, Mr. Macleod Yearsley, Mr. C. A. Ballance, Mr. Scott, Mr. Herbert Tilley (reply)

—— —— of nasal syphilis, Laryng. 14; discussion (p. 14): Mr. Stuart-Low

—— —— to show the result of operative treatment for chronic bilateral empyema of the frontal, ethmoidal and sphenoidal sinuses, Clin. 196

——, discussion on abscess in left cerebral frontal lobe originating from suppuration in left frontal sinus, Laryng. 126

—— —— on bilateral swelling of the septum nasi, Laryng. 94

—— —— on case of chronic suppuration of maxillary antrum, Laryng. 30

—— —— —— of functional aphonia in a male, Laryng. 77

—— —— —— of mucocele of the frontal sinus, Laryng. 71

—— —— —— of nasal obstruction, Laryng. 76

—— —— —— of symmetrical nodules on cords, Laryng. 19

—— —— —— of tuberculous laryngitis treated by thyrotomy, Laryng. 7

—— —— —— of tumour of right thyroid lobe, Laryng. 40

—— —— on cases after radical maxillary antrum operation exemplifying simplification of after-treatment, Laryng. 58

—— —— on effects of long-neglected adenoids on development of upper jaws and nasal septum, Laryng. 85

—— —— on neoplasm of right tonsil, Laryng. 141

—— —— on preparations illustrating diseases of trachea, Laryng. 38

—— —— on proptosis due to distension of frontal sinus, Laryng. 50

—— —— on transverse resection of pharynx with laryngectomy for malignant growth of pharynx, Clin. 68, 71

—— —— on value of X-rays in sounding and washing out frontal sinus, Laryng. 87

——, specimen of a large post-nasal polypus removed from a boy aged 14½, Laryng. 122; discussion (p. 122): Dr. W. Hill, Dr. J. B. Ball, Mr. C. Baber, Dr. W. Williams, Mr. Tilley (reply)

—— —— of subglottic hyperplasia with considerable narrowing of tracheal airway, Laryng. 123; discussion (p. 125): Sir F. Semon

——, stereoscopic slide showing well-marked development of orbito-ethmoidal cells, Laryng. 123

Timothy grass bacillus, loss of acid-fast quality in subcultures of, Path. 73

Tinnitus in labyrinthitis, Otol. 55

TIRARD, N.—Discussion on complications of pneumonia, Med. 43

Tobacco-smoking, effect on blood-pressure, Therap. 36, 37, 38

——, see also *Nicotine*

TOD, HUNTER F.—Case of complete occlusion of the posterior nares with flattening of the nose externally and almost complete absence of the nasal septum, Otol. 133; discussion (p. 133): Dr. McBride, Dr. Grant, Mr. Tod (reply)

——, case of venous angeioma of soft palate, Laryng. 73; discussion (p. 73): Dr. McBride, Dr. Milligan, Dr. FitzGerald Powell, Mr. Waggett, Mr. Steward, Mr. Tod (reply)

—— —— of vertigo, the symptoms pointing to some inflammatory lesion within the semicircular canals, Otol. 28; discussion (p. 29): Mr. A. L. Whitehead, Mr. Scott, Mr. Lake, Mr. West, Dr. McBride, Mr. Tod (reply)

——, discussion on angeioma of right tonsil, Laryng. 109

—— —— on case of primary epithelioma (?) of tympanum, Otol. 35

—— —— on crossed abducens paralysis in a case of cerebellar abscess, Otol. 89

—— —— on operative surgery of labyrinthitis, Otol. 78, 82

—— —— on subcutaneous induration of neck of uncertain origin, Laryng. 113

—— —— on vestibulotomy, Otol. 156

——, lateral sinus thrombosis, subsequent meningitis (meningitis serosa), recovery, Otol. 30

——, two cases of epithelioma of the ear, Otol. 15; discussion (p. 16): Mr. Whitehead, Dr Milligan, Dr. Bronner, Dr. McBride, Mr. Tod (reply)

Toes, clubbing of fingers and toes with caries of spine (P. M. Heath), Clin. 234

TOMKINSON, J. GOODWIN.—Cauterization as an adjuvant to radiotherapy, Electr. 83; discussion (p. 88): Mr. W. Deane Butcher, Dr. David Arthur, Dr. Ross, Dr. W. Ironside Bruce, Dr. J. Goodwin Tomkinson (reply)

Tongue (enlarged), see *Macroglossia* neurofibromatosa

——, epithelioma of, treated by X-rays (W. Ironside Bruce), Electr. 59

—— —— of tongue and fauces, case shown after operations (W. Chichele Nourse), Laryng. 56

——, paralysis of, unilateral (W. Jobson Horne), Laryng. 53

—— and fauces, case of epithelioma of, shown after operations (W. Chichele Nourse), Laryng. 56

Tonic spasm, mechanism of, Med. 96

Tonsil, specimen of, composed of mass of papillomata (A. R. Tweedie), Laryng. 70

——, swelling of (H. B. Jones), Laryng. 143

—— (left), epithelioma of left tonsil, faucial pillar and uvula (J. Dundas Grant), Laryng. 29

—— (right), angeioma of (A. Wylie), Laryng. 109

—— ——, neoplasm of (C. A. Parker), Laryng. 140

—— and larynx, lupus of (J. Dundas Grant), Laryng. 78

Tonsils, normal and hypertrophied pharyngeal, in Southdown sheep: specimens (H. J. Davis), Laryng. 138

Tonsillitis, acute, complicating pneumonia, Med. 14

TOOTH, H. H.—Discussion on acute ulcerative colitis, Med. 6

Torticollis tic, Neur. 44, 45

Tortoise, tonus curves from cardiac auricle of, Med. 254, 255

Torulæ in aural discharges, Otol. 112

TOWNSEND, T. SUTTON.—Discussion on hairball in the stomach, Clin.

Toxæmia as explanation of causation of lymphocythæmia, Clin. 170

Toxic affections of muscles and lower neurones (C. E. Beevor), Neur. 2

—— theory of arteriosclerosis not tenable, Therap. 34, 35

Trachea, diphtheria affecting, preparation illustrating (W. Jobson Horne), Laryng. 37

——, fibrous constriction of (H. Tilley), Laryng. 35

——, papilloma of, preparations illustrating (W. Jobson Horne), Laryng. 37

Tracheal airway, considerable narrowing of, in subglottic hyperplasia (H. Tilley), Laryng. 123

Tracheotomy and galvano-cautery in treatment of extensive tuberculosis of larynx; complete cicatrization (StClair Thomson), Laryng. 9

Traction exerted by pendent limb causing coxa valga, Surg. 125

Trapezium; congenital absence of both thumbs, trapezium and scaphoid (H. Curtis), Clin. 236

Traumatism as an agent in causation of chorea compared with fright and shock, Therap. 72
—— followed by coxa valga, Surg. 111, 118, 133
—— in causation of labyrinthitis, Otol. 50
——, pyosalpinx due to, in case of uterine fibroid, Obst. 123
Treadgold, views on etiology of lymphocythæmia, quoted, Clin. 169, 173
Trees as breeding-places of the mosquito, Epid. 312, 316, 317
Tremor, connection of nystagmus with, Neur. 81
—— of right arm, associated with epilepsy (F. E. Batten), Neur. 54
Treponomema pallida, see *Spirochæta pallida*
TREVES, Sir FREDERICK, Bt., G.C.V.O., C.B.—Preventive medicine at Panama, Epid. 303-313; discussion (p. 313): Dr. Newsholme, Sir Shirley Murphy, Dr. Sandwith, Mr. Malcolm Watson, Professor Ronald Ross, C.B., Sir F. Treves (reply), Colonel Macpherson
TREWBY, H. W.—Case showing incomplete eruption of the temporary molars, Odont. 99; discussion (p. 100): Mr. J. F. Colyer
Trinidad, epidemic of small-pox of irregular type in, during 1902 4, Epid. 229
—— —— ——, age-incidence in, Epid. 242
—— —— ——, race-incidence, Epid. 246
—— —— ——, seasonal incidence, Epid. 247
—— —— ——, sex-incidence, Epid. 246
Trismus (functional) (L. G. Guthrie), Neur. 103
Trochanter (great), flattening in region over, in coxa valga, Surg. 136
Tropical diseases (certain), remedies required for treatment and prophylaxis of (J. Cantlie), Therap. 22
Trousseau on epileptic aura, quoted, Med. 95
Trypanosomes, drugs used to destroy, Therap. 26
——, see also *Sleeping sickness*
TUBBY, A. H.—Case of isolated paresis of right serratus magnus, exhibited by Dr. Ernest Jones, Neur. 9
——, coxa valga (collum valgum), Surg. 107; discussion (p. 141): Mr. Gordon Watson, Mr. Warrington Haward, Mr. Tubby (reply)
Tubercle bacillus (avian) pathogenic to guinea-pig in very limited degree, Path. 70
—— —— (human) not convertible into avian by inoculation into birds, Path. 70, 71
—— —— —— pathogenic to pigeon in very limited degree, Path. 69
—— —— ——, proof of production of endocarditis by, Path. 116, 121, 122
—— —— ——, question of convertibility into the avian, experiments and results, Path. 54-60
—— —— ——, relation to avian, tested by means of "opsonic" index, Path. 60-65
—— —— (human and avian), presentation to normal human leucocytes in serum of patients suffering from tuberculosis, amount of phagocytosis occurring after saturation of serum with human and avian bacillus, Path. 60-65
Tubercle of cervix uteri (P. Horrocks), Obst. 66
—— of one sixth nucleus (E. Farquhar Buzzard), Neur. 38
Tuberculides, cases (J. L. Bunch), Derm. 175
—— (J. H. Sequeira), Derm. 39
Tuberculin cutaneous reaction, Path. 74, 75
—— —— and ophthalmic reactions, non-agreement in results, Path. 76
—— ophthalmic reaction, conclusions respecting its value, Path. 76, 77, 78
—— —— —— experiences with (L. J. Austin and O. Grünbaum), Path. 74
—— —— ——, positive, obtained in case of Bazin's disease (J. M. H. MacLeod), Derm. 31
Tuberculin (T.R), oral administration in caseating pulmonary tuberculosis (A. Latham), Clin. 100
—— —— and horse serum, oral administration, Med. 235, 237
—— —— —— —— —— in case of early pulmonary tuberculosis with tuberculous infection of glands, Med. 203, 204
—— —— —— —— —— —— of tuberculous disease of hip, Med. 207
—— —— —— —— —— in cases of genito-urinary tuberculosis, Med. 247

Tuberculin (T.R.) and horse serum, oral administration in cases of pulmonary tuberculosis, Med. 207-211, 247, 250

—— —— ——, rectal administration in cases of tuberculous peritonitis, Med. 205, 206

—— —— and saline solution, oral administration in case of tuberculous and staphylococcic infection, Med. 203

—— —— and saline solution, or horse serum, oral administration (A. Latham, H. D. Spitta and A. C. Inman), Med. 195-239

—— —— —— —— —— —— ——, diagnostic use of, Med. 216, 217

—— —— —— —— —— —— ——, dosage, Med. 219-223

—— —— —— —— —— —— —— ——, carefulness and judgment required in, Med. 220

—— —— —— —— —— —— —— ——, control by opsonic index, Med. 251

—— —— —— —— —— —— —— ——, temperature and condition of patient a guide to, Med. 220, 221, 322

—— —— —— —— —— —— ——, effects, Med. 213

—— —— —— —— ——, rectal administration, diagnostic use of, Med. 216

Tuberculosis, association with, suggested by histological resemblance of granuloma annulare to sarcoid, Derm. 151

——, clinical symptoms, relation of opsonic index to, Med. 232

—— developing on site of vaccination scar (J. L. Bunch), Derm. 164

——, experimental production of, Path. 108

—— not prevalent among keepers in Zoological Society's Gardens, Path. 28

—— of ear, Otol. 106, 119, 120

—— of Fallopian tube, primary unilateral (H. Macnaughton-Jones), Obst. 177

—— of larynx (W. Hill), Laryng. 64

—— ——, extensive, completely cicatrized, after treatment with galvano-cautery and tracheotomy (StClair Thomson), Laryng. 9

——, presence in families of school children, Epid. 55

——, probably combined with syphilis in larynx (Sir F. Semon), Laryng. 135

—— simulated by parasitic invasion in birds, Path. 21

—— treatment, future lines of, Med. 238

—— (abdominal), commonest form in birds, Path. 17

—— (avian), coexistence of aspergillosis with, Path. 20

—— —— ——, met with chiefly among birds kept in captivity, Path. 22

—— ——, prevalence in Zoological Society's Gardens, Path. 22, 28

—— (avian and human), relationship between (S. G. Shattock, C. G. Seligmann, L. S. Dudgeon, and P. N. Panton), Path. 15-73

—— —— —— —— ——, practical importance owing to consumption of birds as human food, Path. 16

—— (cutaneous), inoculation for, by von Pirquet's method, Path. 74, 75

—— (genital), in women, treatment (F. J. McCann), Obst. 259

—— —— —— ——, conservative, Obst. 261

—— (genito-urinary), treatment by oral administration of tuberculin and horse serum, Med. 247

—— (miliary), diagnosis by Röntgen ray examination subsequently confirmed, Electr. 43

—— ——, inverse temperature of, effect of tuberculin on, Med. 211

—— (pulmonary), diagnosis aided and completed by Röntgen rays, examples, Electr. 38-41

—— ——, prognosis aided by Röntgen rays, Electr. 41

—— ——, ratio of transference of infection, Epid. 214 (footnote)

—— ——, septal deflection occurring in subject of (A. H. Burt), Laryng. 111

—— ——, temperature, relation of increase of, to exercise, Med. 249, 251

—— —— ——, relation of opsonic index to, Med. 224

—— ——, treatment by oral administration of horse serum, Med. 246

—— —— —— by oral administration of tuberculin and horse serum, Med. 207-211, 247

—— —— ——, see also *Phthisis*

—— —— (caseating), treatment by tuberculin (T.R.) and fresh horse serum administered by mouth (A. Latham), Clin. 100

Tuberculosis (pulmonary) (early "closed"), treatment by oral administration of serum and tuberculin, tuberculin and saline solution, Med. 203. 204
——, see *Synovitis*, tuberculous
Tuberculous disease in labyrinthitis, Otol. 53, 59, 65, 77
—— material from birds, feeding of pigeons with, Path. 34
—— —— —— ——, feeding of Rhesus monkey with, Path. 53
—— —— ——, inoculation of guinea-pigs with suspensions of, Path. 50
—— —— —— ——, arrest and retrogression of process, Path. 52
—— organs of birds, salt suspensions of, inoculated into pigeons, experiments and results, Path. 46
—— —— of guinea-pigs infected by means of human sputum, inoculation of pigeons with, Path. 31
—— and staphylococcic infection, treatment by oral administration of serum and staphylococci, tuberculin, and saline solution, Med. 203
——, staphylococcic, and streptococcic infection. treatment by oral administration of staphylococci, streptococci, and horse serum, Med. 202
Tumour, see under names of organs and regions affected; see also *Dermoid tumour*, *Fibroid tumour*, *Intracranial tumour*, *Melanotic tumour*, *Sarcoid tumours*
Tumours consisting of carcinoma and sarcoma. explanation of, Path. 170
——, metastasis of, new views respecting, Surg. 3
Turbinal (inferior); ulceration (malignant growth of), microscopic specimen (E. Cresswell Baber), Laryng. 41
—— (middle), localized pulsating growth of. causing recurrent attacks of dangerous epistaxis (A. Tweedie), Laryng. 1
TURNER, A. LOGAN.—Discussion on cytological examination in discharge in cases of middle ear and maxillary sinus suppuration, Otol. 163
——, patient after operation for left temporo-sphenoidal abscess occurring in the course of a recent middle ear suppuration, Otol. 137, 142. [For discussion see *Permewan*, W.—Case of infection of lateral sinus]
——, patient operated upon for sigmoid sinus thrombosis, Otol. 137, 142. [For discussion see *Permewan*, W.—Case of infection of lateral sinus]
——, two patients after the mastoid operation with preservation of the tympanic membrane and ossicles, Otol. 144, 148. [For discussion see *Barr*, J. Stoddart—Notes on partial grafting . . . in after-treatment of radical mastoid operation]
—— and WADE, HENRY.—Macroscopic and microscopic preparations from a case of right temporo-sphenoidal abscess and left sigmoid sinus thrombosis, Otol. 138, 142. [For discussion see *Permewan*, W., case of infection of lateral sinus]
TURNER, DAWSON.—Some reflections based upon the work done in the Electrical Department of the Royal Infirmary, Edinburgh, Electr. 118
Turner, John, on intravascular clotting as cause of epilepsy, quoted, Med. 93
TURNER, J. G.—Discussion on case of extraction of upper wisdom tooth, Odont. 103
——, the effects of chronic suppuration in the molar of a horse, Odont. 26
——, some clinical notes on pyorrhœa alveolaris, Odont. 104; discussion (p. 105): Mr. Norman Bennett, Mr. Kenneth Goadby, Mr. J. G. Turner (reply)
TURNER, W., CARGILL, L. V., and THOMSON, STCLAIR.—Case of abscess in the left cerebral frontal lobe, originating from suppuration in the left frontal sinus, Laryng. 125. [For discussion see *Cargill*, L. V.]
Turner, W. Aldren, on petit mal, quoted, Med. 99
TURNEY, H. G.—Case of syringomyelia, Neur. 89; discussion (p. 90): Dr. Head
TWEEDIE, A. R.—A microscopic specimen of a localized pulsating growth of the middle turbinate body which gave rise to recurrent attacks of dangerous epistaxis, Laryng. 1; discussion (p. 1): Dr. Pegler, Dr. de Havilland Hall, Dr. Scanes Spicer, Dr. McKenzie, Dr. Donelan, Mr. Atwood Thorne, Mr. Tweedie (reply)
——, specimen of a tonsil apparently composed of a mass of papillomata, Laryng. 70; discussion (p. 70): Dr. StClair Thomson

TWEEDY, E. HASTINGS. Discussion on the supports of the pelvic viscera, Obst. 220
—— and Gibson, M. J., cases of pubiotomy, mentioned, Obst. 22
Tympanic cavity, primary epithelioma of, on left side, with secondary infection of skin over mastoid region (H. Tod), Otol. 15
—— epithelium in aural discharges, Otol. 99
—— membrane, preservation of, after mastoid operation (A. Logan Turner), Otol. 144
Tympanum, epithelioma, ? primary, following chronic suppurative otitis media (A. L. Whitehead), Otol. 34
——, incision of, Otol. 118
Typhoid fever complicated by purpura haemorrhagica, Clin. 122
—— —— epidemic at Ottweiler, Epid. 209
—— —— —— at Wittlich, Epid. 208
—— ——, etiology, increase in rainfall and consequent floods, how far likely, Epid. 173, 174
—— —— ——, pollution of food, Epid. 171, 172, 187, 188
—— —— ——, pollution of water supplies, Epid. 169-175, 191, 222; see also under *Typhoid fever* (transmission)
—— ——, etiology and epidemiology of, discussion on, Epid. 169-228; introductory address and opening papers (pp. 169-218): E. C. Seaton, D. S. Davies and I. Walker Hall, H Meredith Richards and J. A. H. Brincker, W. H. Hamer; discussion (p. 218-228): Dr. Newsholme, Dr. E. W. Goodall, Dr. C. J. Martin, F.R.S., Colonel Firth, Dr. Franklin Parsons, Dr. Wheaton, Dr. Andrewes, Sir Shirley Murphy, Colonel Davies, Major Horrocks, Dr. Prausnitz, Dr. Butler, Dr. Bond, Dr. Davies (reply), Professor Walker Hall (reply), Drs. Richards and Hamer (reply)
—— ——, gall-bladder lesions rare during attacks of, Epid. 219
—— —— incidence at Metz, Epid. 206-209
—— ——, inoculation against, Epid. 223
—— —— in Surrey, causes of prevalence various, Epid. 171, 172, 173
—— ——, outbreak at Brentry Certified Inebriate Reformatory, Bristol, due to "carrier," Epid. 177
—— —— —— —— —— —— ——, simultaneous outbreaks of swine fever, Epid. 185
—— —— —— at Fleetwood (Militia battalion), cause of, Epid. 222
—— —— —— at Grove House Home for Girls, Brislington, near Bristol, due to "carrier," Epid. 185
—— ——, prevalence in autumn independent of nature of water-supply and amount of rainfall, Epid. 173
—— ——, regional immunity from, Epid. 206-209
—— ——, relation of *Bacillus typhosus* to, Epid. 221, 224
—— —— —— —— —— (W. H. Hamer), Epid. 204-218
—— —— —— —— ——, difficulties respecting, Epid. 215-218, 223
——, transmission by bedding and clothing, Epid. 222
—— —— —— by carriers of *Bacillus typhosus*, Epid. 175-191, 209-214, 225, 226, 227
—— —— —— —— —— ——, see also *Carriers*
—— —— —— by contact with patients, Epid. 223, 227
—— —— —— by milk, through agency of "carrier," Epid. 180
—— —— ——, control observations necessary, Epid. 214
—— —— ——, house incidence theory untenable, Epid. 205, 206
Typhus fever, factors promoting, and factors tending to extinction, Epid. 31
—— —— in Ireland, Epid. 5
—— —— —— contrasted with phthisis, Epid. 20
—— —— —— illustrating relation of poverty to disease, Epid. 1

Ulcer, see also *Rodent ulcer*; and under names of organs and regions
Ulcers, factitious, case for diagnosis (W. S. Fox), Derm. 70
Unconsciousness of epileptic fit, cause of, Med. 95
United States, medical officers of health of, superior status accorded to, Epid. 314

University College Hospital, statistics of complications of pneumonia at, 1897-1906 (S. A. Owen), Med. 70
Uræmia, causation of cerebral manifestations of, Med. 101
——, coma of, Med. 104
——, convulsions of, Med. 105
——, treatment by lumbar puncture, Med. 103
Uratic deposits in gout, and bone changes, Clin. 230
Ureter, see *Calculus*, ureteral
Urine, ammonia in, Path. 140, 141
——, analyses of, in acute yellow atrophy, Path, 141, 142
—— ——, in pregnancy, Path. 136, 137
——, condition of, in cases of lymphocythæmia, Clin. 160
——, incontinence of, treatment by hypnotism, Therap. 56
——, lactic acid in, in eclampsia, Path. 131
—— ——, in epileptics, Path. 132
——, nitrogen in, Path. 131, 133-143
Urotropine, valuable in septic diseases, Obst. 118
Urticaria pigmentosa, case (H. G. Adamson), Derm. 63
—— —— in adult (E. G. Graham Little), Derm. 165
Uterus bicornis duplex, with unilateral hæmatometra (T. Wilson), Obst. 70
—— —— —— —— ——, removal by operation, Obst. 71
—— —— —— —— ——, report on specimen, Obst. 72
——, cancer of, microscopic sections showing cell changes, Obst. 153
—— ——, perithelioma of ovary coexisting with (A. H. N. Lewers), Obst. 156
—— ——, sarcomatous ovarian tumour coexisting with (A. H. N. Lewers), Obst. 69
—— —— of body of, caseating (H. R. Spencer), Obst. 119
——, chorionepithelioma of, with extensive vaginal and visceral metastases (T. W. Eden), Obst. 13
——, fibromyoma of, enucleation during seventh lunar month of pregnancy (H. Williamson), Obst. 73
—— —— (sarcoma) of (J. Bland-Sutton), Obst. 233
——, fibroid tumour of, with pyosalpinx due to traumatism, Obst. 123
—— —— ——, showing sarcomatous degeneration (W. C. Swayne), Obst. 151
—— —— —— —— ——, report of Pathology Committee on, Obst. 151
——, inversion of, chronic, treatment by abdominal hysterotomy (F. W. N. Haultain), Obst. 279, 283, 286
—— —— —— —— by continued elastic pressure (Aveling's method), Obst. 283, 285, 288-290
—— —— —— —— by posterior colpotomy and incision of entire uterus, Obst. 283, 286
—— —— —— —— by taxis, Obst. 282, 283, 285
——, malposition in, cause of multiple dislocations, including congenital dislocation of hips, in child aged 2 weeks (T. H. Openshaw), Clin. 106
——, myoma of, complicated by hæmatosalpinx (H. Macnaughton Jones), Obst. 122
—— occupying sac of inguinal hernia in case of hermaphroditism (T. H. Kellock), Clin. 111
——, pregnant, tolerance of surgical intervention, Obst. 131
—— ——, with fibroid, the latter in state of "red degeneration" (C. E. Purslow), Obst. 42
——, prolapse of, Obst. 218, 219, 223
—— ——, causes, Obst. 224
—— ——, cured by induction of pelvic cellulitis, Obst. 46
——, retraction (contraction) ring of, Obst. 144-146, 148, 149, 150
——, rupture of, in presence of fibroids, Obst. 131, 137, 138
——, sarcoma of, case (C. Lockyer), Obst. 245
—— ——, rarity of, Obst. 233
——, specimen of, removed by Wertheim's method (A. H. N. Lewers), Obst. 100
—— ——, report on (R. D. Maxwell), Obst. 102
——, supports of, Obst. 216

Uterus, suspension of, Obst. 44
—— with fibroids, retroversion of, followed by hæmatosalpinx with acute symptoms (A. Doran), Obst. 174
——, see also *Cervix uteri* ; *Ligaments*, uterine ; *Womb stone*
Uvula, epithelioma of left tonsil, faucial pillar and uvula (J. Dundas Grant), Laryng. 20

Vaccination, question of conferrence of immunity to small-pox by, Epid. 286, 287
——, results of performance during desquamation or shortly after recovery from small-pox, Epid. 289
——, small-pox following recent performance of, Epid. 284
——, table showing state of, among school children, Epid. 50, 51
—— scar, tuberculosis developing on site of (J. L. Bunch), Derm. 164
Vaccines, injection of, in treatment of pneumonia, Med. 15, 16-20, 45
—— —— ——, successful in cases of malignant endocarditis, Med. 15
——, oral administration, dosage, Med. 223
—— —— ——, effect of, Med. 219
—— —— ——, value in treatment of acne, Med. 243
—— —— ——, together with (*a*) normal saline solution, (*b*) fresh horse serum (A. Latham, H. D. Spitta and A. C. Inman), Med. 195-239
Vagal and vaso-vagal attacks in epilepsy, Med. 84
Vagina, atresia of, in adult, acquired (H. Macnaughton Jones), Obst. 325
——, septic discharges from, treatment by ionic medication, Electr. 133
——, stenosis of, three cases of Cæsarean hysterectomy for (K. Das), Obst. 188
——, traumatic atresia of, treated by Cæsarean hysterectomy (A. Routh), Obst. 1
——, tumours of, presence of pigmentation in, indicates high degree of malignancy, Obst. 127
Vagrancy and mendicancy in Ireland after 1847, Epid. 14
Vagus, peripheral end of, results of stimulation, Med. 84
Valve tube, value of, as rectifier, Electr. 98
Variations, acquired, Odont. 110
——, genetic, Odont. 110
Varus nodulosus, Brooke, case (E. G. Graham Little), Derm. 85
Vascular system, dissemination of malignant growths along, Surg. 1
—— ——, growth of malignant disease in (Professor E. Goldmann), Surg. 1
Vaseline, mixture of, with coarse barium of sulphate for shielding parts of scalp not to be exposed in treatment of ringworm by X-rays (A. Whitfield), Derm. 61
Vasomotor changes in splenic polycythæmia, Clin. 42, 43
—— spasm in brain as cause of epilepsy, Med. 87
—— system, instability of, in epilepsy, Med. 82
Vegetable charcoal, in treatment of dyspepsia due to irritable stomach, Therap. 17
—— food, cooking of, untoward effects on teeth and digestion, Therap. 48
—— ——, proportion of, increased under era of [illegible], Therap. 46, 47
Vein (femoral), excision of parts of femoral artery and vein for recurrent sarcoma (C. H. Fagge), Clin. 231
—— ——, common, excision of, with aneurysm of common femoral artery (C. H. Fagge), Clin. 232
—— (jugular), ligature of, in case of infection of lateral sinus ; recovery (W. Permewan), Otol. 139, 140, 142
Venezuela, prevalence of small-pox in villages of, in 1902, Epid. 231, 232
Venous angeioma of soft palate, case (H. Tod), Laryng. 73
Ventricle, fourth, of brain, angio-sarcoma of, Neur. 27
—— —— ——, tumour of choroid plexus of, Neur. 30
—— —— —— —— —— ——, with secondary growths on spinal cord, Neur. 28
—— —— ——, tumours growing on : symptomatology (T. Grainger Stewart), Neur. 26
"Ventricular rhythm," nature of, Therap. 30
VERNON, H. M.—Written communication to discussion on Mendelism in relation to disease, Epid. 161

Verrucose nævus, case of (W. S. Fox), Derm. 12
Version, value of, as means of delivery after craniotomy, Obst. 148
Vertebræ (cervical), disease of, case with pressure symptoms (E. F. Buzzard), Neur. 12
——, unusual deformity of, with seventh cervical ribs (E. F. Buzzard), Neur. 69
Vertex presentations, site for pubiotomy incision in, Obst. 22, 23
Vertigo (aural), cases (R. Lake), Otol. 24; (H. Tod), Otol. 28
——, ablation of both vestibules for relief of (G. Gibson and R. Lake), Otol. 150, 156
—— accompanying vestibular or labyrinthine nystagmus, Neur. 76, 80
—— in labyrinthitis, Otol. 55, 80
Vestibule, aural, description of, Otol. 38
—— ——, ablation of, for relief of vertigo (G. Gibson and R. Lake), Otol. 150, 156
Vestibulotomy, patient exhibited after (J. S. Fraser), Otol. 149
——, double, in labyrinthitis, Otol. 61
—— —— —— ——, with partial removal of cochlea, Otol. 64
——, inferior, in labyrinthitis, Otol. 59
——, superior, in labyrinthitis, Otol. 57
VINRACE, DENNIS.—Discussion on angeioma of right tonsil, Laryng. 109
——, discussion on case of laryngeal paralysis, Laryng. 107
—— —— on subcutaneous induration of neck of uncertain origin, Laryng. 113
Violet light in treatment of lichen planus verrucosus (H. Radcliffe Crocker and G. Pernet), Derm. 38
Virchow, his approval of term "ossific diathesis," Path. 106
Virgil, quotation from, respecting method of raising bees from putrefying carcass of ox, Path. 15
Viscera, transport of bacteria to, Path. 108
—— (abdominal and thoracic), matting of, in avian tuberculosis, Path. 20
—— (pelvic), supports of (W. E. Fothergill), Obst. 43
—— —— —— —— (R. H. Paramore), Obst. 195
—— —— —— —— complexity of, Obst. 204, 205, 222, 225
Vision, results of tests among school-children, Epid. 55
Vocal cord, paralysis of, first symptoms of aneurysm of arch of aorta, Electr. 52
—— —— (left), cancer of, removal of cord (C. J. Symonds), Clin. 185
—— —— ——, epithelioma of (H. Tilley), Laryng. 60
—— —— ——, fibroma of (œdematous) (L. H. Pegler), Laryng. 8, 29
—— —— —— ——, patient shown after removal (C. Horsford), Laryng. 24
—— —— ——, growth on (H. J. Davis), Laryng. 96
—— —— ——, intrinsic neoplasm of, in man aged 76 (R. H. Scanes Spicer), Laryng. 89
—— —— ——, removal of, case showing improved condition of voice after (C. J. Symonds), Clin. 185
—— —— ——, tumour of (L. H. Pegler), Laryng. 8
—— —— (right), congestion of (H. Tilley), Laryng. 18
—— —— —— infiltration of (J. Donelan), Laryng. 83
—— —— ——, loss of movement in case of swelling of right arytænoid (W. H. Kelson), Laryng. 108
—— —— ——, paresis of (D. McKenzie), Laryng. 2
Vocal cords, functional paresis of palate and (E. A. Peters), Laryng. 11
—— ——, symmetrical neoplasms on (W. Jobson Horne), Laryng. 13
—— —— nodules on (H. Barwell), Laryng. 18
—— —— and palate, paralysis of, in case of tabes (C. M. H. Howell), Neur. 66
Voice, improved condition of, after excision of larynx (C. J. Symonds), Clin. 184
—— —— —— ——, after removal of one vocal cord (left) (C. J. Symonds), Clin. 185
——, see also *Baritone voice*
Volition, loss of, producing automatic action of spinal centres, Neur. 72, 73
——, quicker motion in direction of, feature of acquired nystagmus, Neur. 77
——, tics of, Neur. 42, 43
——, see also *Will power*

Vomiting in labyrinthitis, Otol. 55, 82
——, severe, of pregnancy, connection with yellow atrophy of liver, Path. 133
—— —— ——, high coefficient of ammonia in urine, Path. 133
Vulva, melanotic tumour of (E. Holland), Obst. 124
—— —— ——, irregularity of pigmentation in, cases, Obst. 126
—— —— ——, rarity of occurrence, Obst. 126
——, tumours of, presence of pigmentation indicates high degree of malignancy, Obst. 127

Wade, Henry, and Turner, A. Logan.—Macroscopic and microscopic preparations from a case of right temporo-sphenoidal abscess and left sigmoid sinus thrombosis, Otol. 138, 143. [For discussion see *Permewan*, W.—Case of infection of lateral sinus
Wadsworth, on immunity against effects of pneumococcus, alluded to, Med. 4
Waggett, E. B.—Case of multiple telangiectases, Laryng. 70; discussion (p. 70): Mr. De Santi, Dr. Watson Williams
——, discussion on case for diagnosis, Otol. 27
—— —— —— of mucocele of frontal sinus, Laryng. 72
—— —— —— of primary epithelioma (?) of tympanum, Otol. 35
—— —— —— of ulceration and destruction of epiglottis, Laryng. 34
—— —— —— of venous angeioma of the soft palate, Laryng. 73
—— —— on keloid following mastoid operation, Otol. 22
—— —— on malignant growth of septum and naso-pharynx removed by external osteoplastic operation, Laryng. 118
—— —— on new instrument for intralaryngeal operations, Laryng. 25
—— —— on operative surgery of labyrinthitis, Otol. 77
—— —— on pulsating growth in left external auditory meatus, Otol. 33
—— —— on sudden deafness in case of syphilis, Otol. 9
Wagner, case of voidance *per rectum* of larvæ of *Eristalis arbustorum*, alluded to, Path. 151
Waldeyer, on the supports of the pelvic viscera, quoted, Obst. 56
Walker, C. E.—The influence of the testis upon the secondary sexual characters of fowls (from the Cancer Research Laboratories, University of Liverpool), Path. 153
Wall, R. Cecil.—Discussion on rheumatism and chorea in childhood, Therap. 79, 86
——, discussion on therapeutics of indigestion, Therap. 20
Wallace, G. B., researches of, on nutmeg poisoning detailed, Therap. 40, 41, 42
Wallace, J. Sim.—Classification of dento-facial irregularities, Odont. 140; discussion (p. 144): Mr. G. Northcroft, Dr. Baker, Mr. Schelling, Mr. D. Gabell, Mr. Rushton, Mr. Baldwin, Mr. J. F. Colyer, Dr. Sim Wallace (reply)
——, discussion on heredity and dental disease, Odont. 119
—— —— on movements of mandible, Odont. 97
—— —— on treatment of children from dental aspect, Odont. 73
Wallis, Frederick C.—Discussion on ionic medication, Electr. 77
—— and Bruce, W. Ironside.—The treatment of ulcerative proctitis by ionic cataphoresis, Surg. 176; discussion (p. 184): Mr. Warrington Haward, Dr. Curtis Webb, Dr. H. K. Ramsden, Mr. Lockhart Mummery, Mr. Swinford Edwards, Mr. Wallis (reply), Dr. Ironside Bruce (reply)
Walsh, David.—Case of fibromata of skin with developing neurofibromata, Derm. 189
——, case of primary cutaneous carcinoma of the chest involving the neighbouring glandular areas, Derm. 187
——, discussion on case of X-ray dermatitis, Derm. 177
——, spurious acromegaly in a patient suffering from exophthalmic goitre associated with a congenitally high forehead, Clin. 195
Warburg's tincture, superiority over quinine in treatment of malaria, Therap. 24
Ward, E.—Case of subcutaneous induration of the neck of uncertain causation (introduced by Mr. Hunter Tod), Laryng. 112; discussion (p. 112): Dr. Grant, Mr. Clayton Fox, Dr. Vinrace, Dr. Syme, Mr. Tod, Dr. FitzGerald Powell, Dr. Ward (reply)
Warts, treatment by magnesium ions, Electr. 75

Warts (cutaneous), causing confusion in X-ray diagnosis of calculi, Electr. 29

—— (gonorrhœal) on penis, treatment by X-rays (A. D. Reid), Electr. 63

—— (senile), developing into fungating growths, Derm. 56

Wash-leather colour of mucous membrane in catarrhal ulcerative proctitis, Surg. 176, 177

Water-cress, ingestion of, containing ova of *Eristalis tenax*, probable cause of presence of larvæ in bowel, Path. 148, 149

Water-supplies from wells in chalk, pollution of, cause of spread of typhoid fever, Epid. 191, 224

——, isolation of *Bacillus typhosus* from, Epid. 225

——, pollution of, cause of spread of typhoid fever, Epid. 169-175, 191, 222

—— —— —— —— —— ——, over-due importance attached to doctrine, Epid. 170-174

——, provision at Colon and City of Panama, Epid. 309

Watson, C. Gordon.—Discussion on treatment of coxa valga, Surg. 141

Watson, Malcolm.—Discussion on preventive medicine at Panama, Epid. 315

de Watteville current, uses of, Electr. 106, 109

Watts, Stephen, remarks on performance of arterio-venous anastomosis, quoted, Clin. 206

Webb, J. Curtis. - Discussion on sciatica and its surgical treatment, Surg. 174

——, discussion on treatment of ulcerative proctitis by zinc cataphoresis, Surg. 184

Weber, F. Parkes.—Arteritis obliterans of the lower extremity with intermittent claudication ("angina cruris"), Clin. 44

——, case of angina pectoris with aortitis, Clin. 114; discussion (p. 117): Dr. James Mackenzie, Dr. F. de Havilland Hall, Sir Dyce Duckworth, Dr. Garrod, Sir. T. Barlow, Dr. Parkes Weber (reply)

—— —— of multiple hereditary developmental angeiomata (telangiectases) of the skin and mucous membranes associated with recurring hæmorrhages, Laryng. 43

—— —— of multiple xanthoma of the face of the diabetic type in an infant, Derm. 92; discussion (p. 93): Dr. Radcliffe Crocker

——, discussion on case of ascites for which paracentesis had been repeatedly performed in one year, Clin. 192

—— —— on case of caseating pulmonary tuberculosis treated by tuberculin (T.R.) and fresh horse serum, both administered by the mouth, Clin. 103

—— —— on case of enlarged spleen with polycythæmia, without cyanosis, Clin. 61

—— —— —— of leucodermia with symptoms of Addison's disease, Clin. 212

—— —— —— of myxœdema with optic atrophy, Clin. 195

—— —— —— of myxœdema with unusual features, Clin. 29

—— —— —— of segmental pan-hypertrophy, Neur. 11

—— —— —— of slowly progressive hemiplegia, Neur. 6

—— —— —— of unusual situation of gouty deposit, Clin. 55

—— —— on acute ulcerative colitis, Med. 165

—— —— on arteritis obliterans of right upper extremity associated with malformation of both clavicles, Neur. 102

—— —— on congenital heart disease in adults, Clin. 37

—— —— on lymphangioma of face, Clin. 92

—— —— on lymphangioplasty for brawny arm of breast cancer, Clin. 189

—— —— lymphocythæmia, Clin. 178

—— —— on ochronosis, Clin. 58

—— —— on ruptured aortic valve, Clin. 99

—— —— on splenic polycythæmia with cyanosis, Clin. 42

—— —— —— —— without cyanosis, Clin. 61

—— —— on treatment of pneumothorax with aspiration under X-rays, Clin. 39

——, hæmangiectatic hypertrophy of the foot, possibly of spinal origin, Clin. 193, Derm. 59, Neur. 49

——, intermittent claudication in a lower extremity (angina pedis or angina cruris) from chronic arteritis obliterans, Neur. 50; discussion (p. 51): Dr. Head, Sir W. Gowers, Dr. Parkes Weber (reply)

Weber, F. Parkes.—Multiple hereditary developmental angeiomata (telangiectases) of the skin and mucous membranes, with recurring epistaxis, Clin. 65; discussion (p. 66): Dr. C. O. Hawthorne, Sir T. Barlow, Mr. Stephen Mayou, Dr. Parkes Weber (reply)
——, multiple idiopathic hæmorrhagic sarcoma of the skin, Clin. 235
——, œdema of hands and feet with mediastinal affection, Clin. 192
——, spurious (?) acromegaly, Clin. 104
—— and Michels, E.—Ischæmic disturbance of the right upper extremity, Neur. 100
Webster, A., on association of arrest of pulse with epileptic and convulsive attacks, quoted, Med. 74
Weichselbaum's bacillus, see *Diplococcus intracellularis*
Weights and measurements of school children, Epid. 49, 50
Weil, on effect of hypodermic administration of horse serum and fresh antidiphtheritic serum on the blood, quoted, Med. 198
Weismann, diagrammatic explanation of his theory of heredity, Odont. 111
——, views on variations in germ cells and somatic cells, quoted, Odont. 110
Weiss on the causation of Raynaud's disease, quoted, Med. 88
Wertheim's method of hysterectomy in removal of cancerous uterus and glands (C. H. Roberts), Obst. 43
—— —— —— for advanced carcinoma of cervix (A. H. N. Lewers), Obst. 100
West, C. E.—Discussion on case for diagnosis, Otol. 27
——, discussion on foreign body in ear, Otol. 7
—— —— on case of primary epithelioma (?) of tympanum, Otol. 35
—— —— on cases of vertigo (Ménière's disease), Otol. 26, 30
—— —— on keloid following mastoid operation, Otol. 21
—— —— on mechanism of nystagmus, Neur. 80
——, specimens of the temporal bone illustrating operations on the labyrinth, Otol. 91
——, and Scott, Sydney R.—The operative surgery of labyrinthitis based upon an experience of thirty cases (with bibliography), Otol. 37-73; discussion (p. 75): Dr. W. Milligan, Dr. Bronner, Mr. Waggett, Mr. Yearsley, Dr. FitzGerald Powell, Dr. Dundas Grant, Dr. Albert Gray, Mr. Hunter Tod, Mr. R. Lake, Mr. Fagge, Dr. Kelson, Dr. Paterson, Dr. McBride, Dr. Donelan, Mr. West (reply), Mr. Scott (reply)
West, Samuel.—Discussion on acute ulcerative colitis, Med. 166
——, discussion on complications of pneumonia, Med. 41
—— —— on diagnostic value of Röntgen rays in some diseases of the chest, Electr. 51
—— —— on multiple rheumatic nodules in adult, Clin. 14
—— —— on pyo-pericardium cured by drainage, Clin. 16
——, the acute suffocative catarrh of Laennec and other conditions from which it should be distinguished; a typical case associated with a peculiar bacillus, Med. 151; discussion (p. 158): Dr. Gee, Dr. Wethered, Dr. William Ewart, Dr. West (reply)
Westmacott, F. H.—Discussion on bilateral sarcoma of upper maxillæ, Laryng. 22
——, discussion on bony outgrowths from maxilla and mandible, Laryng. 95
—— —— on case of chronic suppuration of maxillary antrum, Laryng. 31
—— —— on the effects of long-neglected adenoids on development of upper jaws and nasal septum, Laryng. 85
—— —— on value of X-rays in sounding and washing out frontal sinus, Laryng. 87
Westminster Hospital, statistics of complications of pneumonia at (1897-1900) (A. M. Gossage), Med. 61, 66
Wethered, F. J.—Discussion on acute suffocative catarrh of Laennec, Med. 159
Whait, J. R.—Case of amyotonia congenita, Neur. 61; discussion (p. 61): Dr. F. E. Batten, Dr. Head, Dr. Beevor
Wheat, immunity to disease in, experiments respecting, Epid. 140
Wheaton, S. W.—Discussion on etiology and epidemiology of typhoid fever, Epid. 223
Whistle, see *Edelmann's whistle*; *Galton's whistle*
White, W. Hale.—Discussion on ochronosis, Clin. 58
White, W., his case of maggots passed after taking purgative, quoted, Path. 150

WHITEHEAD, A. L.—Case of primary epithelioma (?) of the tympanum following chronic suppurative otitis media, Otol. 34; discussion (p. 35): Mr. Waggett, Dr. Milligan, Mr. Tod, Mr. Cheatle

——, discussion on bilateral sarcoma of upper maxillæ, Laryng. 23

—— —— on case for diagnosis, Otol. 27

—— —— —— of stenosis of right auditory meatus, Otol. 14

—— —— —— of vertigo, Otol. 29

—— —— on cases of epithelioma of ear. Otol. 16

—— —— on lateral sinus thrombosis, Otol. 139

—— —— on mastoid operation, Otol. 147

—— —— on sudden deafness in case of syphilis, Otol. 9

—— —— on unusual case of cerebellar abscess, Otol. 23

—— —— on vestibulotomy, Otol. 156

WHITFIELD, ARTHUR.—Case of disseminated lupus with chronic lymphangitis of the face and osteoarthritis of the hands, Derm. 93; discussion (p. 94): Dr. Radcliffe Crocker, Sir Malcolm Morris, Dr. Whitfield (reply)

—— case of macular atrophy of the scalp (pseudo-pelade of Brocq), Derm. 60

——, discussion on case of bromide eruption, Derm. 75

—— —— —— of chronic X-ray dermatitis of hands, Derm. 80

—— —— —— of (?) dermatitis artefacta, Derm. 85

—— —— —— of lupus with unusual features, Derm. 78

—— —— on cases of elephantiasis græcorum, Derm. 56

—— —— on dermatitis herpetiformis of unusual type, Derm. 180

—— —— on molluscum contagiosum, Derm. 172

—— —— on pityriasis rosea, Derm. 183

—— —— on well-marked rosacea, associated with phlyctenular conjunctivitis and ulceration of the eyes, Derm. 181

——, long-standing pruritus in a woman aged 22, Derm. 22

——, microscopical specimen and pure culture of a yeast derived from a case of intertriginous dermatitis of the cruro-scrotal region, Derm. 190

——, new substance for shielding those parts of the scalp which it is not wished to expose in the treatment of ringworm by means of the X-rays, Derm. 61

——, rodent ulcer treated by the introduction of zinc ions, Derm. 21

——, specimens from case of syphilis, Derm. 172; discussion (p. 174): Dr. Crocker

Whooping-cough, changes in age- and sex-incidence before and during school life, Epid. 58

Wilbrand, H., his explanation of mechanism of nystagmus, Neur. 79

WILLETT, J. ABERNETHY.—Case of "womb stone" (exhibited for J. Preston Maxwell), Obst. 228

WILLIAMS, A. WINKELRIED.—Case of lupus erythematosus saccharo-mycetiform, Derm. 94; discussion (p. 94): Dr. Radcliffe Crocker

——, case of rodent ulcer of the ala nasi in a man aged 36, Derm. 62

——, sections of paraffinoma, Derm. 23

WILLIAMS, C. THEODORE.—Discussion on case of caseating pulmonary tuberculosis treated by tuberculin (T.R.) and fresh horse serum, both administered by the mouth, Clin. 103

——, discussion on ruptured aortic valve, Clin. 99

—— —— on tumour of mediastinum, Clin. 95

WILLIAMS, H. LLOYD.—Discussion on movements of mandible, Odont. 97

Williams, J. Whitridge, on vomiting in pregnancy, quoted, Path. 133

WILLIAMS, P. WATSON.—Case of laryngeal disease, Laryng. 119; discussion (p. 120): Sir F. Semon, Dr. Jobson Horne, Dr. Pegler, Mr. De Santi, Dr. Watson Williams (reply)

——, case of laryngeal ulceration, Laryng. 23; discussion (p. 23): Sir F. Semon, Mr. Barwell, Dr. Smurthwaite, Dr. Watson Williams (reply)

——, case of malignant growth of the septum and naso-pharynx removed by an external osteoplastic operation, Laryng. 115; discussion (p. 118): Mr. De Santi, Dr. P. McBride, Mr. Stuart-Low, Mr. C. Nourse, Mr. Waggett, Dr. Watson Williams (reply)

WILLIAMS, P. WATSON.—Discussion on bilateral swelling of the septum nasi, Laryng. 93
——, discussion on case of chronic suppuration of maxillary antrum, Laryng. 31
—— —— —— of multiple telangiectases, Laryng. 71
—— —— on fistulous opening in middle line of neck, Laryng. 3
—— —— on four cases illustrating surgery of sphenoidal sinus, Laryng. 120
—— —— on radiograph of nasal cavities, Laryng. 52
—— —— on specimen of large post-nasal polypus removed from boy aged 14½, Laryng. 123
—— —— on subglottic laryngeal tumour, Laryng. 90
—— —— on tuberculosis and syphilis in larynx, Laryng. 136
—— —— on value of X-rays in sounding and washing out frontal sinus, Laryng. 87
——, microscopic sections and drawings illustrating the pathogenesis of some forms of nasal polypi, Laryng. 52
——, photograph showing remarkable development of the orbito-ethmoidal cells, Laryng. 121; discussion (p. 121): Dr. Jobson Horne, Dr. Paterson, Dr. Watson Williams (reply)
——, series of skiagrams showing the accessory sinuses of the nose, Laryng. 121
——, universal laryngeal forceps for use by the direct and indirect methods, Laryng. 53
WILLIAMSON, HERBERT.—Enucleation during the seventh lunar month of pregnancy of a uterine fibromyoma weighing 17½ lb.; premature labour two days later, Obst. 73; discussion (p. 77): Dr. Herbert Spencer, Dr. James Oliver, Mrs. Boyd, Dr. Amand Routh, Dr. Macnaughton Jones, Dr. Maxwell, Dr. Williamson (reply)
——, and HOLLAND, EARDLEY L.—Case of intra-uterine death of the fœtus occurring in six consecutive pregnancies, with observations upon the importance of the examination of the fœtal tissues for the presence of the *Spirochæta pallida*, Obst. 161; discussion (p. 170): Dr. Herbert Spencer, Dr. Amand Routh, Dr. Maxwell, Dr. Beckett-Overy
Will-power, development of, essential under hypnotic treatment, Therap. 60
——, see also *Volition*
WILSON, S. A. K.—Case of segmental pan-hypertrophy, Neur. 11; discussion (p. 11): Dr. Parkes Weber
——, discussion on case of disease of cervical vertebræ with pressure symptoms, Neur. 14
—— —— —— of nerve-root grafting, Neur. 8
—— —— on cases of myatonia congenita and of toxic affection of muscles and lower neurones, Neur. 3
—— —— on tumour of fourth ventricle, Neur. 37
WILSON, THOMAS.—Pubiotomy, with notes of an illustrative case, Obst. 19; discussion (p. 29): Dr. H. Spencer, Dr. Macnaughton Jones, Dr. Griffith, Dr. C. H. Roberts, Dr. Blacker, Dr. Wilson (reply)
——, unilateral hæmatometra removed by operation, Obst. 41; discussion (p. 41): Dr. Peter Horrocks, Dr. Griffith, Dr. Thomas Wilson (reply)
——, uterus bicornis duplex with unilateral hæmatometra, Obst. 70
—— —— —— —— —— ——, report on specimen, Obst. 72
WILTSHIRE, H. W., and BRISCOE, J. CHARLTON.—Statistics of complications of pneumonia from King's College Hospital, 1897-1906, Med. 69
WINGRAVE, V. WYATT.—Discussion on infiltration of the nasal cavities, Laryng. 92
——, pathological report on case in which an endothelioma had been removed from the hard palate, Laryng. 62
——, pathological report of case of complete closure of the anterior nares with partial and progressive atresia of the naso-pharynx and oro-pharynx, Laryng. 57
——, pathological report on case of epithelioma of the tongue and fauces shown after operations, Laryng. 56
——, pathological report on Mr. W. Chichele Nourse's microscopic sections of sarcoma of nose, Laryng. 55
——, pathological report on specimen of epithelioma of tongue and fauces, Laryng. 56
——, the clinical pathology of aural discharges, with references, Otol. 95-117; discussion (p. 118): Dr. Jobson Horne, Dr. Wingrave (reply), Dr. Milligan, Dr. Kelson, Dr. Grant, Dr. Dan McKenzie, Dr. McBride, Dr. Wingrave (reply)

Winter, on suspension of uterus, quoted, Obst. 44
Wisdom tooth, difficult extraction of (A. W. W. Baker), Odont. 130
—— —— invaded by bone, in case of difficult extraction, Odont. 135
—— —— (upper), extraction, case of (W. Rushton), Odont. 101
Withering, W., on administration of digitalis, quoted, Therap. 85
Wittlich, typhoid fever epidemic at, Epid. 208
Wolf and Ewing, analyses of urine in pregnancy, Path, 134, 136, 142
"Womb stone," case of (exhibited by J. Abernethy Willett for J. Preston Maxwell), Obst. 228
Women, bacteriuria in, treatment by ionic medication, Electr. 135
——, genital tuberculosis in, treatment (F. J. McCann), Obst. 259
—— most frequent "carriers" of *Bacillus typhosus*, Epid. 177, 212
——, pelvic disease in, treatment by ionic medication in obstinate cases, Electr. 126-138
—— —— ——, treatment by ionic medication in milder cases gratifying, Electr. 136
——, rarity of true angina pectoris among, Clin. 118
Wound, dressing of, in compound and ununited fractures, Surg. 221, 236
—— infection, prevention in operation for goitre, Surg. 32
WORRALL, E. S.—Discussion on electrical treatment of atonic conditions of digestive system, Electr. 110
——, discussion on ionic medication, Electr. 77
Wright, Sir A. E., his method of taking opsonic index, alluded to, Med. 178, 189, 190
—— on treatment of infective diseases by vaccines, alluded to, Med. 195
WYLIE, ANDREW.—Case of angeioma of the right tonsil, Laryng. 109; discussion (p. 109): Dr. Vinrace, Mr. Hunter Tod, Dr. Horsford (reply)
——, case of swelling of arytænoid, Laryng. 60
WYNTER, W. ESSEX.—Amyotonia congenita, Clin. 48
——, cure of ascites by permanent drainage through the femoral ring, Clin. 49; discussion (p. 50): Mr. Sampson Handley
——, methæmoglobinæmia of twelve years' standing, Clin. 48; discussion (p. 48): Dr. Poynton, Sir T. Barlow

Xantho-erythrodermia perstans ? case of (H. Radcliffe Crocker and G. Pernet), Derm. 36-38
Xanthoma of face, multiple, diabetic type, in infant (F. Parkes Weber), Derm. 92
X-rays, see *Röntgen* rays

Yaws, see *Frambœsia*
YEARSLEY, P. MACLEOD.—Case of suppuration in the right labyrinth; operation, recovery, Otol. 92
——, discussion on branchial sinus leading into external auditory meatus, Otol. 91
—— —— on case of chronic eczema of both auricles and meatuses, Otol. 132
—— —— —— of unilateral nerve deafness, Otol. 130
—— —— on deafness resulting from epidemic cerebrospinal meningitis, Otol. 87
—— —— on keloid following mastoid operation, Otol. 22
—— —— on operative surgery of labyrinthitis, Otol. 77
—— —— on specimens and photographs of pathological conditions found in the labyrinth, Otol. 93
—— —— on unilateral hysterical nerve deafness, Otol. 128
—— microscopic specimen and drawing of a foreign body, Otol. 6; discussion (p. 7): Mr. C. E. West, Mr. W. H. Bowen
——, specimens of necrosis of the labyrinth, Otol. 131
Yeast derived from case of intertriginous dermatitis of cruro-scrotal region (A. Whitfield) Derm. 190
Yellow atrophy of liver, connection of severe vomiting of pregnancy with, Path. 133
—— —— ——, excretion of urine in, Path. 142

Yellow fever, cause of unhealthiness and mortality at Isthmus of Panama, Epid. 304
—— ——, mortality of British Army from, in West Indies (1780-96), Epid. 304, 305
—— ——, preventive measures against, instituted by Isthmian Canal Commission, Epid. 311
Young, J. K., description of his cases of coxa valga, quoted, Surg. 111
——, description of museum specimens of coxa valga, quoted, Surg. 115
YULE, UDNY.—Discussion on Mendelism in relation to disease, Epid. 164
Yunnan (South China), plague always endemic at, Epid. 92
—— —— ——, starting-point of modern epidemics of plague, Epid. 92

Zinc ions in treatment of lupus, Electr. 75
—— —— —— of rodent ulcer, Electr. 73; (A. Whitfield), Derm. 21
—— —— —— of ulceration of cornea, Electr. 75
—— —— —— —— of rectum, Electr. 75
—— ——, introduction of, in treatment of ulcerative proctitis, Surg. 176-178
—— —— —— —— —— ——, method, Surg. 178
—— —— —— —— —— ——, record of cases treated, Surg. 179
—— —— —— —— ——, use of cocaine preceding, Surg. 184
Zoological Society's Gardens, prevalence of avian tuberculosis in, Path. 22, 28
—— —— ——, tuberculosis not prevalent among keepers, Path. 28
Zweifel on acidosis in pregnancy, Path. 131
Zygote, definition and nature of, Epid. 136-138

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